objective findings. Thus, recognizing, analyzing, and properly interpreting skin lesions are the sine qua non of dermatologic diagnosis.

PHYSICAL EXAMINATION

Appearance Uncomfortable, "toxic," well

Vital Signs Pulse, respiration, temperature

Skin: "Learning to Read" The entire skin should be inspected and this should include mucous membranes, genital and anal regions, as well as hair and nails and peripheral lymph nodes. Reading the skin is like reading a text. The basic skin lesions are like the letters of the alphabet: their shape, color, margination, and other features combined will lead to words, and their localization and distribution to a sentence or paragraph. The prerequisite of dermatologic diagnosis is thus the recognition of (1) the type of skin lesion, (2) the color, (3) margination, (4) consistency, (5) shape, (6) arrangement, and (7) distribution of lesions.

Recognizing Letters: Types of Skin Lesions

- **Macule** (Latin: *macula*,"spot") A macule is a circumscribed area of change in skin color without elevation or depression. It is thus not palpable. Macules can be well- and ill defined. Macules may be of any size or color (Image I-1). White, as in vitiligo; brown, as in café-au-lait spots; blue, as in Mongolian spots; or red, as in permanent vascular abnormalities such a port-wine stains or capillary dilatation due to inflammation (erythema). Pressure of a glass slide (*diascopy*) on the border of a red lesion detects the extravasation of red blood cells. If the redness remains under pressure from the slide, the lesion is purpuric, that is, results from extravasated red blood cells; if the redness disappears, the lesion is due to vascular dilatation. A rash consisting of macules is called a *macular exanthem*.
- **Papule** (Latin: *papula*, "pimple") A papule is a superficial, elevated, solid lesion, generally considered <0.5 cm in diameter. Most of it is elevated above, rather than deep within, the plane of the surrounding skin (Image I-2). A papule is palpable. It may be well- or ill defined. In papules the elevation is caused by metabolic or locally produced deposits, by localized cellular infiltrates, inflammatory or noninflammatory, or by hyperplasia of local cellular elements. Superficial papules are sharply defined. Deeper dermal papules have indistinct borders. Papules may be dome-shaped, cone-shaped or flat-topped (as in lichen planus) or consist of multiple, small, closely packed, projected elevations that are known as a *vegetation* (Image I-2). A rash consisting of papules is called a papular *exanthem*. Papular exanthems may be grouped ("lichenoid") or disseminated (dispersed). Confluence of papules leads to the development of larger, usually flat-topped, circumscribed, plateau-like elevations known as plaques (French: *plaque*, "plate"). See below.

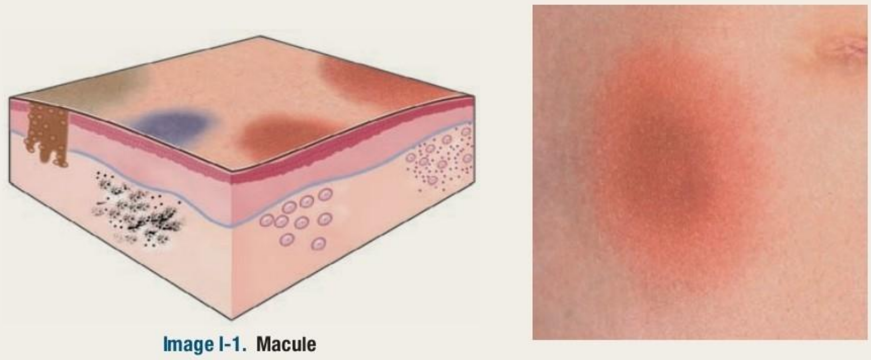


Image I-1. Macule

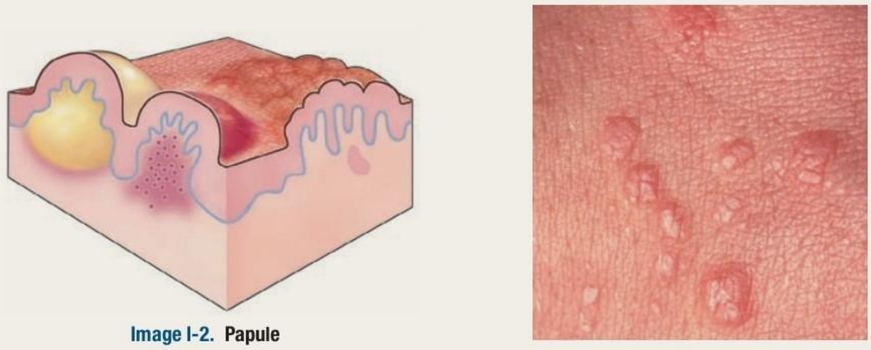
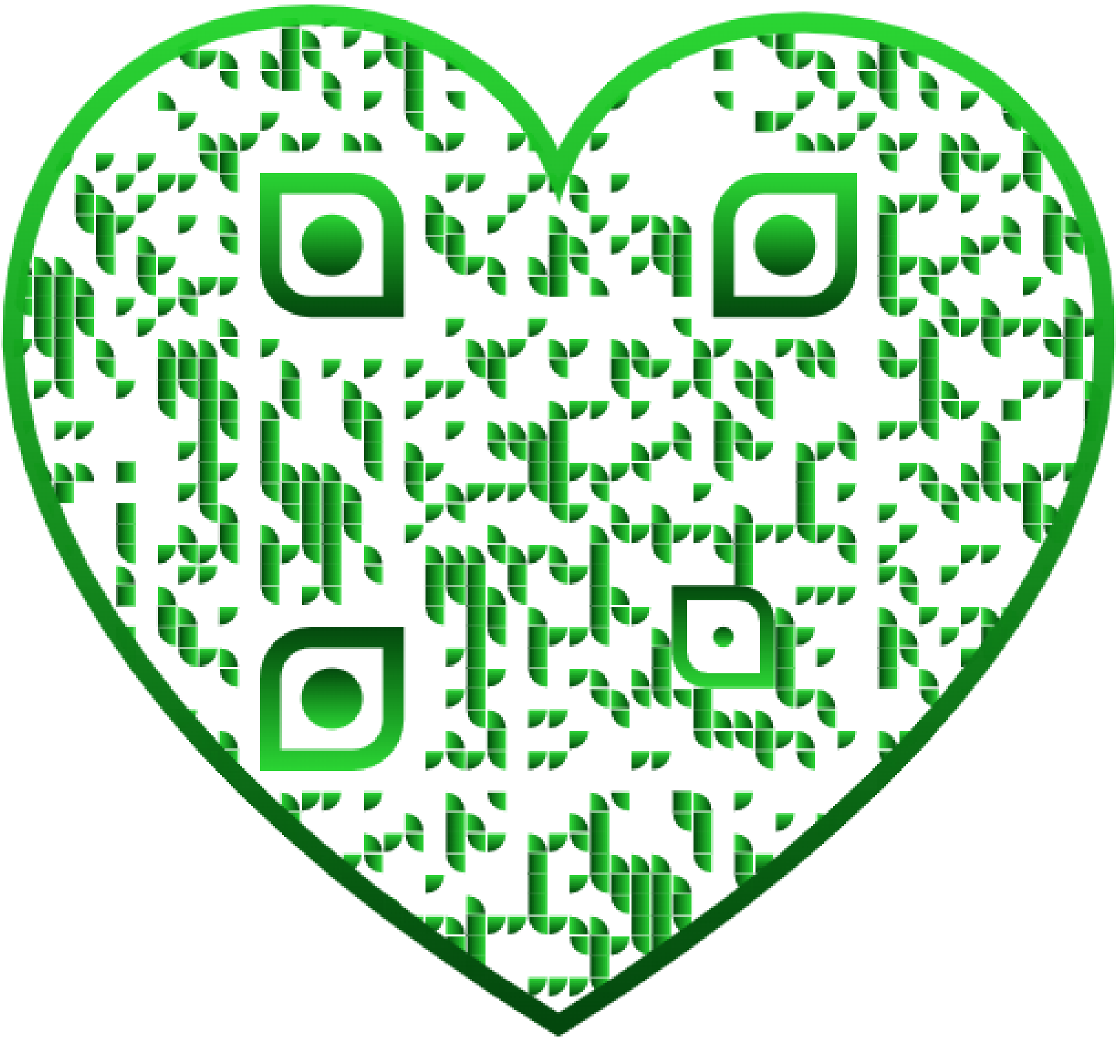


Image I-2. Papule

- **Plaque** A plaque is a plateau-like elevation above the skin surface that occupies a relatively large surface area in comparison with its height above the skin (Image I-3). It is usually well defined. Frequently it is formed by a confluence of papules, as in psoriasis. *Lichenification* is a less well defined large plaque where the skin appears thickened and the skin markings are accentuated. Lichenification occurs in atopic dermatitis, eczematous dermatitis, psoriasis, lichen simplex chronicus, and mycosis fungoides. A *patch* is a barely elevated plaque—a lesion fitting between a macule and a plaque—as in parapsoriasis or Kaposi sarcoma.
- **Nodule** (Latin: *nodulus*, "small knot") A nodule is a palpable, solid, round, or ellipsoidal lesion that is larger than a papule (Image I-4) and may involve the epidermis, dermis, or subcutaneous tissue. The depth of involvement and the size differentiate a nodule from a papule. Nodules result from inflammatory infiltrates, neoplasms, or metabolic deposits in the dermis or subcutaneous tissue. Nodules may be well defined (superficial) or ill defined (deep); if localized in the subcutaneous tissue, they can often be better felt than seen. Nodules can be hard or



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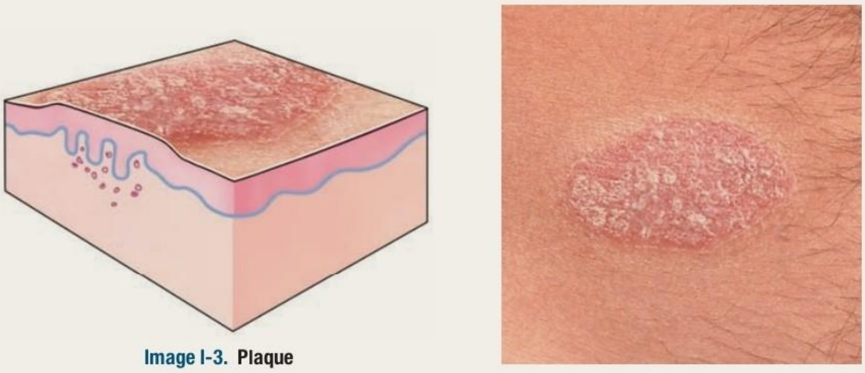

Image I-3. Plaque

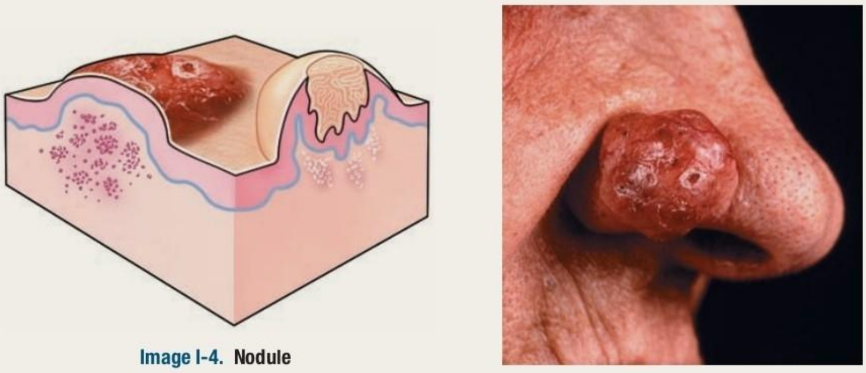

Image I-4. Nodule

- **Wheal** A wheal is a rounded or flat-topped, pale red papule or plaque that is characteristically evanescent, disappearing within 24–48 h (Image I-5). It is due to edema in the papillary body of the dermis. Wheals may be round, gyrate, or irregular with pseudopods—changing rapidly in size and shape due to shifting papillary edema. A rash consisting of wheals is called a *urticarial exanthema* or *urticaria*.
- **Vesicle-Bulla** (*Blister*) (L[illegible] *vesicula*, "little bladder"; *bulla*, "bubble") A vesicle (<0.5 cm) or a bulla (>0.5 cm) is a circumscribed, elevated, superficial cavity containing fluid (Image I-6). Vesicles are dome-shaped (as in contact dermatitis, dermatitis herpetiformis), umbilicated (as in herpes simplex), or flaccid (as in pemphigus). Often the roof of a vesicle/bulla is so thin that it is transparent, and the serum or blood in the cavity can be seen. Vesicles containing serum are yellowish; those containing blood from red to black. Vesicles and bullae arise from a cleavage at various levels of the superficial skin; the cleavage may be subcorneal or within the visible epidermis (i.e., intraepidermal vesication) or at the epidermal–dermal i[illegible]ce (i.e., sub), as in Image I-6. Since ve[illegible]ullae are always sup[illegible]cial they are a[illegible] well defined. A rash consisting of vesi[illegible] called a *vesicular exanthem*; a rash consist[illegible] of bullae a *bullous exanthem*.

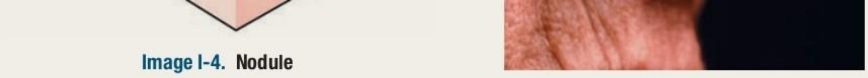

Image I-4. **Nodule**

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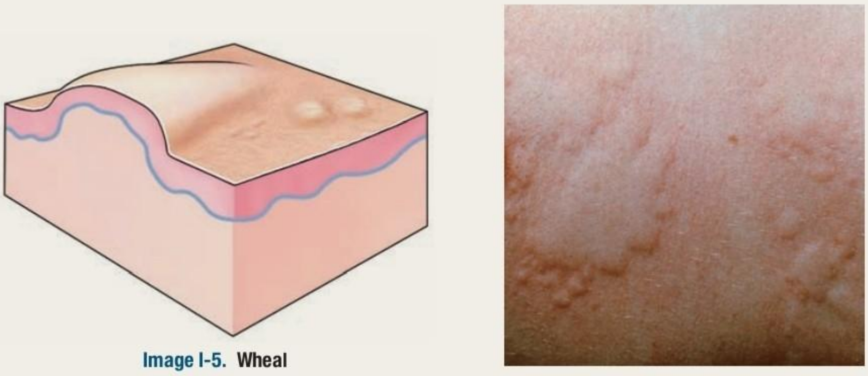


Image I-5. **Wheal**

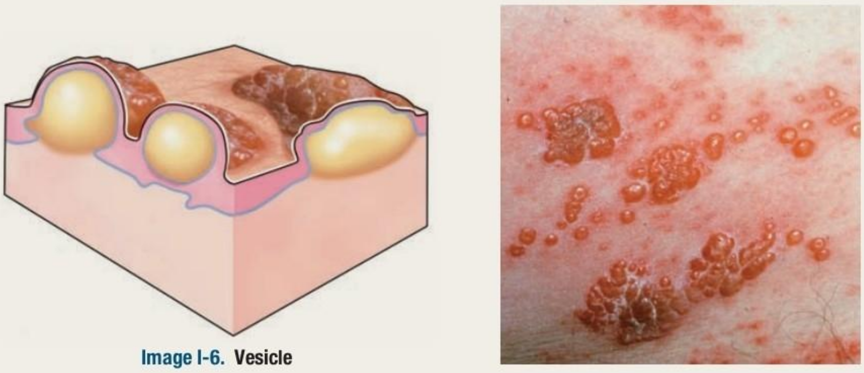
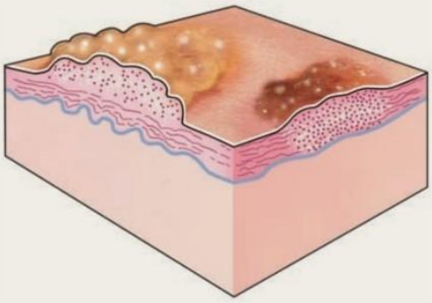


Image I-6. **Vesicle**

- **Pustule** (Latin: *pustula*, "pustule") A pustule is a circumscribed superficial cavity of the skin that contains a purulent exudate (Image I-7), which may be white, yellow, greenish-yellow, or hemorrhagic. Pustules thus differ from vesicles in that they are not clear but have a turbid content. This process may arise in a hair follicle or independently. Pustules may vary in size and shape. Pustules

colored, delicate, glistening particulates on the surface and are typically found in impetigo. When the exudate involves the entire epidermis, the crusts may be thick and adherent, and if it is accompanied by necrosis of the deeper tissues (e.g., the dermis), the condition is known as *ecthyma*.

- **Scales (squames)** (Latin: *squama*, "scale") Scales



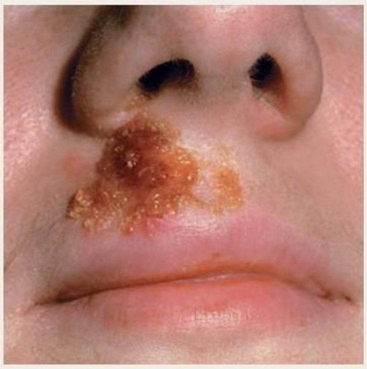
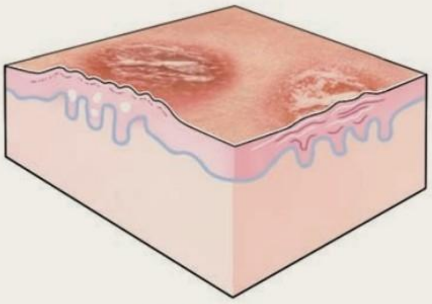


Image I-8. **Crust**



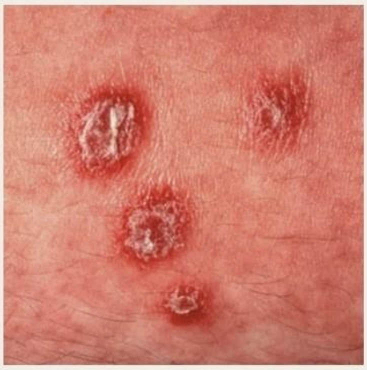
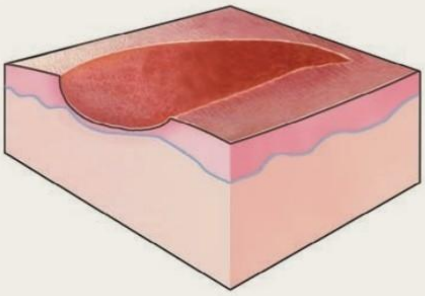


Image I-9. **Scale**

the result of intraepidermal or subepidermal cleavage and thus of vesicles or bullae.

- **Ulcer** (Latin: *ulcus*, "sore") An ulcer is a skin defect that extends into the dermis or deeper (Image I-11) into the subcutis and always occurs within pathologically altered tissue. An ulcer is therefore always a secondary phenomenon. The pathologically altered tissue giving rise to an ulcer is usually seen at the border or the base of the ulcer and is helpful in determining its cause. Other features helpful in this respect are whether borders are elevated, undermined, hard, or soggy; location of the ulcer; discharge; and any associated topographic features, such as nodules, excoriations, varicosities, hair distribution, presence or absence of sweating, and arterial pulses. Ulcers always heal with scar formation.



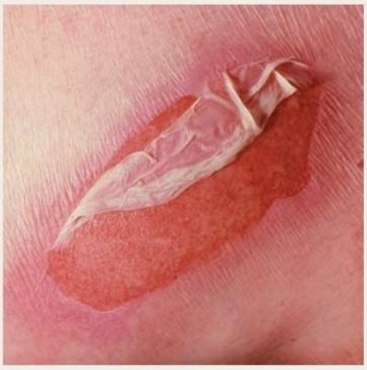
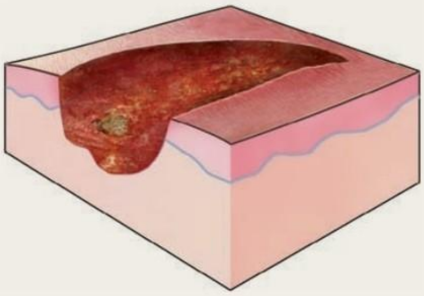


Image I-10. **Erosion**



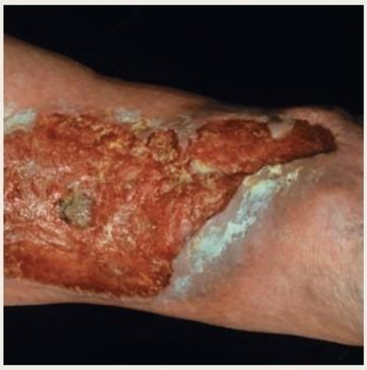
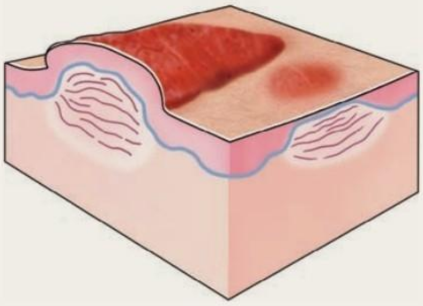


Image I-11. **Ulcer**

Image I-11. Ulcer

- **Scar** A scar is the fibrous tissue replacement of the tissue defect by previous ulcer or a wound. Scars can be hypertrophic and hard (Image I-12) or atrophic and soft with a thinning or loss of all tissue compartments of the skin (Image I-12).
- **Atrophy** This refers to a diminution of some or all layers of the skin (Image I-13). Epidermal atrophy is manifested by a thinning of the epidermis, which becomes transparent, revealing the papillary and subpapillary vessels; there are loss of skin texture and cigarette paper-like wrinkling. In dermal atrophy, there are loss of connective tissue of the dermis and depression of the lesion (Image I-13).
- **Cyst** A cyst is a cavity containing liquid or solid or semisolid (Image I-14) materials and may be superficial or deep. Visually it appears like a spherical, most often dome-shaped papule or nodule, but upon palpation it is resilient. It is lined by an epithelium and often has a fibrous capsule; depending on its contents it may be skin colored, yellow, red, or blue. An epidermal cyst producing keratinaceous material and a pilar cyst that is lined by a multilayered epithelium are shown in Image I-14.



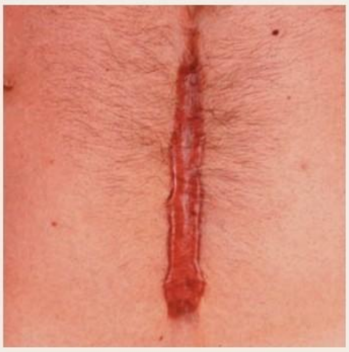
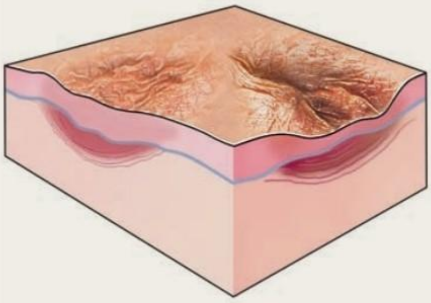


Image I-12. Scar



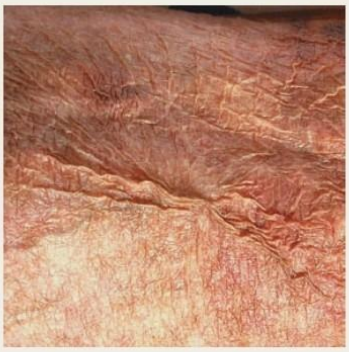
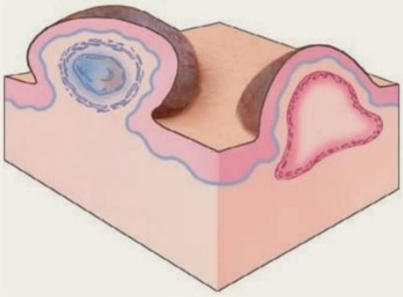


Image I-13. Atrophy



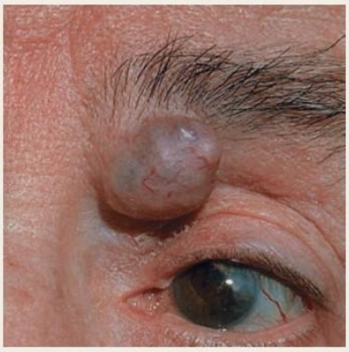


Image I-14. Cyst

Shaping Letters into Words: Further Characterization of Identified Lesions

- **Color** Pink, red, purple (purpuric lesions do not blanch with pressure with a glass slide [diascopy]), white, tan, brown, black, blue, gray, and yellow. The color can be uniform or variegated.
- **Margination** Well (can be traced with the tip of a pencil) and ill defined.
- **Shape** Round, oval, polygonal, polycyclic,

- "Chronic illness" syndrome: fatigue, weakness, anorexia, weight loss, and malaise

2. **History of skin lesions. Seven key questions:**
   - When? Onset
   - Where? Site of onset
   - Does it itch or hurt? Symptoms
   - How has it spread (pattern of spread)? Evolution
   - How have individual lesions changed?

sebum, lipids, fatty acids, keratin, bacteria enter the dermis, provoking an inflammatory and foreign-body response. Intense inflammation leads to scars.

Contributory Factors. Acnegenic mineral oils, rarely dioxin, and others.

tracts (Fig. 1-4). *Sinuses*: draining epithelial-lined tracts, usually with nodular acne. *Scars*: atrophic depressed (often pitted) or hypertrophic (at times, keloidal). *Seborrhea* of the face and scalp often present and sometimes severe.

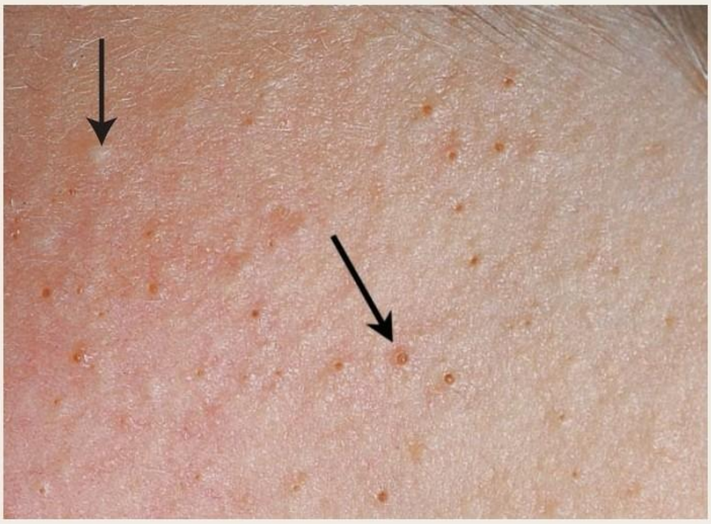


Figure 1-1. Acne vulgaris: comedones Comedones are keratin plugs that form within follicular ostia, frequently associated with surrounding erythema and pustule formation. Comedones associated with small ostia are referred to as closed comedones or “white heads” (upper arrow); those associated with large ostia are referred to as open comedones or “black heads” (lower arrow). Comedones are best treated with topical retinoids.

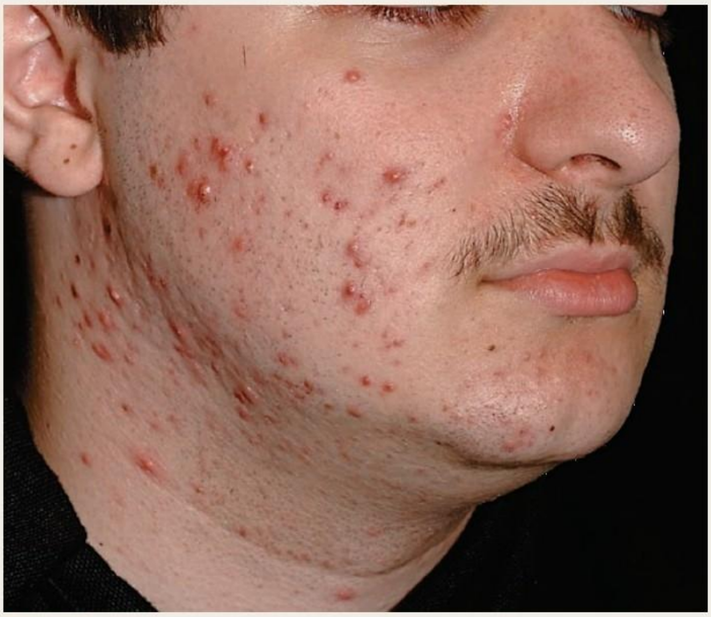
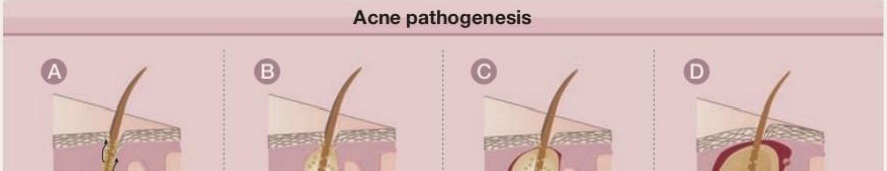


Figure 1-2. 20-year-old male In this case of papulopustular acne, some inflammatory papules become nodular and thus represent early stages of nodulocystic acne.



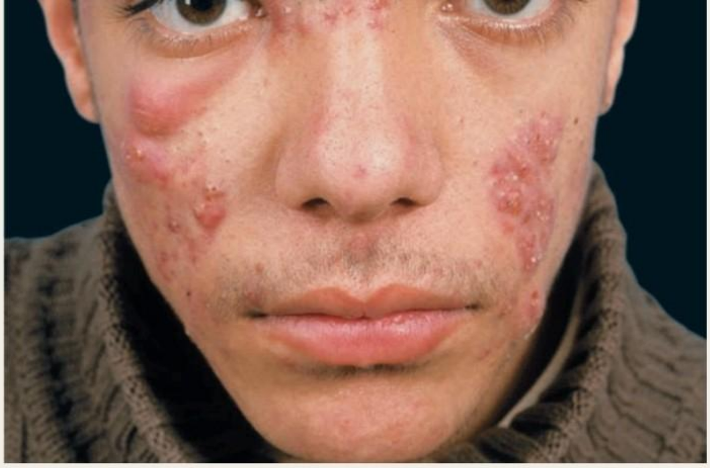


Figure 1-4. Nodulocystic acne A symmetric distribution in the face of a teenage boy. This image clearly shows that even nodulocystic acne starts with comedones—both open and closed comedones can be seen in this face—that then transform into papulopustular lesions, which enlarge and coalesce eventually to lead to nodulocystic acne. It is not surprising that these lesions are very painful, and it is understandable that nodulocystic acne also severely impacts the social life of these adolescents.

epidermal growth factor inhibitors (see Section 23), halogenated compounds.

Acne Aestivalis. No comedones. Papular eruption after sun exposure. Usually on forehead, shoulders, arms, neck, and chest.

Gram-Negative Folliculitis. Multiple tiny yellow pustules on top of acne vulgaris in long-term antibiotic administration.

Diagnosis and Differential Diagnosis

Note: Comedones are required for diagnosis of any type of acne. Comedones are not a feature of acne-like conditions (above), and the following conditions: **Face** *S. aureus* folliculitis, pseudofolliculitis barbae, rosacea, perioral dermatitis. **Trunk** *Malassezia* folliculitis, "hot-tub" pseudomonas folliculitis, *S. aureus* folliculitis, and acne-like conditions (see above).

Laboratory Examination

No laboratory examinations are required. In the overwhelming majority of acne patients, hormone levels are normal. If an endocrine disorder is suspected, determine free testosterone, follicle-stimulating hormone, luteinizing hormone, and DHEAS to exclude hyperandrogenism and polycystic ovary syndrome. Recalcitrant acne can also be related to congenital adrenal hyperplasia (11β or 21β hydroxylase deficiency). If systemic isotretinoin treatment is planned, determine transaminase (ALT, AST), triglyceride, and cholesterol levels.

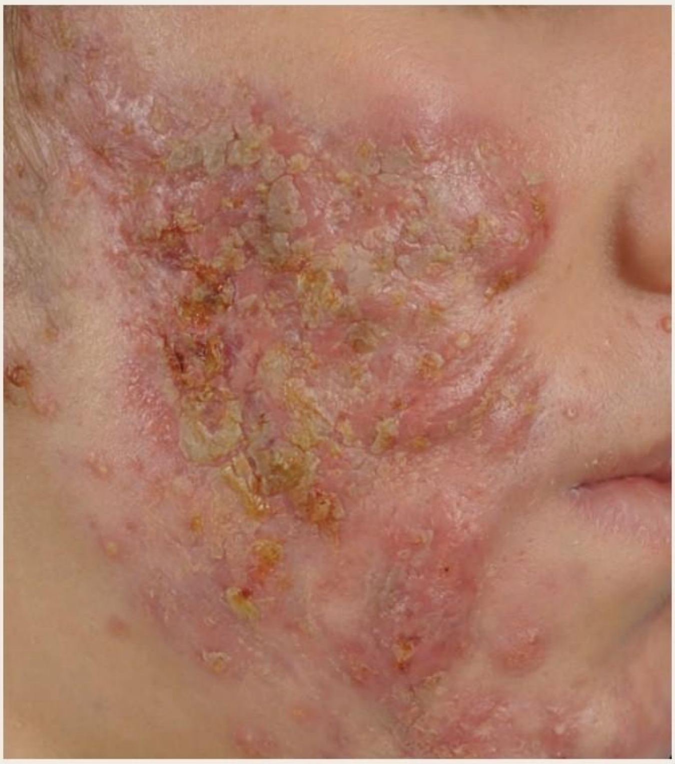


Figure 1-5. Acne conglobata In this severe nodulocystic acne, there are large confluent nodules and cysts forming linear mounds that correspond to interconnecting channels. There is pustulation, crusting, and scarring and lesions are very painful.

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Course

Often clears spontaneously by the early twenties but can persist to the fourth decade or older. Flares occur in the winter and with the onset of menses. The sequela of acne is scarring that may be avoided by treatment, *especially with oral isotretinoin early in the course of the disease* (see below).

Management

The goal of therapy is to remove the plugging of the pilar drainage, reduce sebum production, and treat bacterial colonization. Long-term goal is prevention of scarring.

Mild Acne

Use topical antibiotics (clindamycin and erythromycin) and benzoyl peroxide gels (2%, 5%, or 10%). Topical retinoids (retinoic acid, adapalene, tazarotene) require detailed instructions regarding gradual increases in concentration from 0.01% to 0.025% to 0.05% cream/gel or liquid.

Best combined with benzoyl peroxide–erythromycin gels.

Note: Acne surgery (extractions of comedones) is helpful only when properly done and after pretreatment with topical retinoids.

Moderate Acne. Add oral antibiotics to the above regimen. Minocycline is most effective, 50–100 mg/d, or doxycycline, 50–100 mg twice daily, tapered to 50 mg/d as acne lessens. Use of oral isotretinoin in moderate acne to prevent scarring has become much more common and is effective.

Severe Acne. In addition to topical treatment, systemic treatment with isotretinoin is indicated for cystic or conglobate acne or for any other acne refractory to treatment. This retinoid inhibits sebaceous gland function and keratinization and is very effective. Oral isotretinoin leads to complete remission in almost all cases, which last for months to years in the majority of patients.

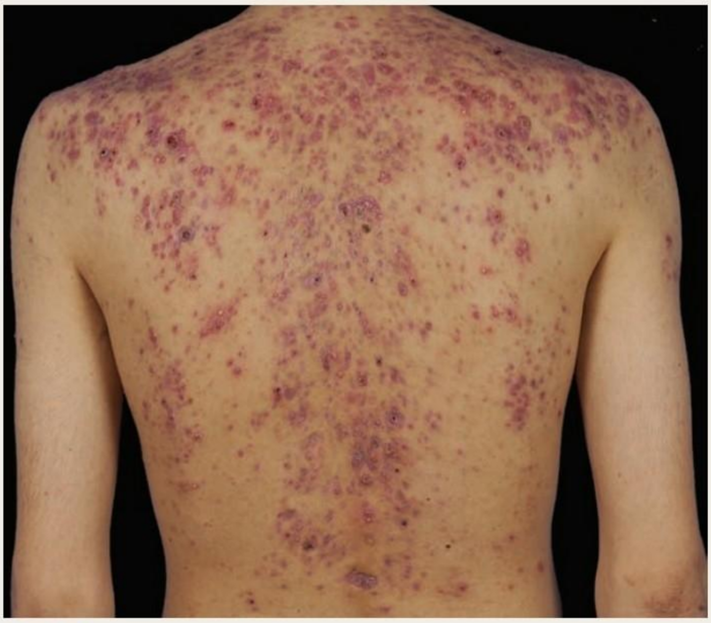

Figure 1-6. Acne conglobata on the trunk Inflammatory nodules and cysts have coalesced, forming abscesses and even leading to ulceration. There are many recent red scars following resolution of inflammatory lesions on the entire back but also on the chest.

Indications for Oral Isotretinoin. Moderate, recalcitrant, nodular acne.

Contraindications. Isotretinoin is teratogenic and effective contraception is imperative. Concurrent tetracycline and isotretinoin may cause pseudotumor cerebri (benign intracranial swelling); therefore, the two medications should *never* be used together.

Warnings. Determine blood lipids, transaminases (ALT, AST) before therapy. About 25% of patients can develop *increased plasma triglycerides*. Patients may develop mild-to-moderate elevation of transaminase levels that normalize low potency (class III) topical glucocorticoids. Dry lips and cheilitis almost always occur and must be treated. Reversible thinning of hair may occur very rarely, as may paronychia. *Nose*: Dryness of nasal mucosa and nosebleeds occur rarely. *Other systems*: Rarely, depression, headaches, arthritis, and muscular pain, pancreatitis occur. For additional rare possible complications, consult the package insert.

Dosage. Isotretinoin, 0.5–1 mg/kg given in divided doses with food. Most patients clear within 20 weeks with 1 mg/kg. Recent studies suggest that 0.5 mg/kg is equally effective.

Other Systemic Treatments for Severe Acne. Adjunctive systemic glucocorticoids may be required in severe acne conglobata, acne fulminans, and the SAPHO and PAPA syndromes. The TNF-α inhibitor infliximab and anakinra are investigational drugs in these severe forms alcohol; exposure to sun and heat. Acne may have preceded the onset of rosacea by years but rosacea usually arises de novo.

Duration of Lesions. Days, weeks, months.

Skin Symptoms. Concern about cosmetic facial appearance.

Skin Lesions. **Early.** Pathognomonic flushing—"red face" (Fig. 1-7); tiny papules and papulopustules (2–3 mm), pustule often small (≤1 mm) and on the apex of the papule (Figs. 1-8 and 1-9). *No comedones*.

hyperplasia, and epithelioid granuloma without caseation.

Differential Diagnosis

Facial Papules/Pustules. Acne (in rosacea there are no comedones), perioral dermatitis, *S. aureus* folliculitis, gram-negative folliculitis, *D. folliculorum* infestation.

Facial Flushing/Erythema. Seborrheic dermatitis, prolonged use of topical glucocorticoids, systemic lupus erythematosus; dermatomyositis.

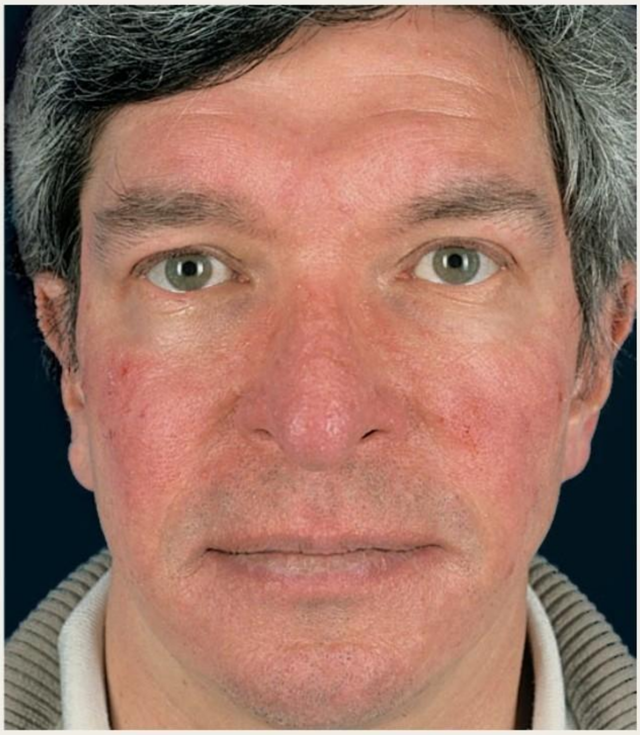

Figure 1-7. Erythematous rosacea (stage I) The early stages of rosacea often present by episodic erythema, "flushing and blushing," which is followed by persistent erythema, which is due to multiple tiny telangiectasias, resulting in a red face.

Course

Prolonged. Recurrences are common. After a few years, the disease may disappear spontaneously; usually it is for lifetime. Men and very rarely women may develop rhinophyma, gnathophyma, etc.

Management

Prevention. Marked reduction or elimination of alcohol may be helpful in some patients.

Topical

- *Metronidazole gel* or *cream*, 0.75% or 1%, once or twice daily.
- *Topical antibiotics* (e.g., erythromycin gel) less effective.

Systemic. Oral antibiotics are more effective than topical treatment.

- *Minocycline or doxycycline*, 50–100 mg once or twice daily, first-line antibiotics; very effective. *Tetracycline*, 1–1.5 g/d in divided doses until clear; then gradually reduce to once-daily doses of 250–500 mg; oral metronidazole 500 mg bid, effective.

Maintenance Treatment. Minocycline or doxycycline 50 mg/d of 50 mg on alternate days or 250–500 g tetracycline.

Oral Isotretinoin. For severe disease (especially stage III) not responding to antibiotics and topical treatments. A low-dose regimen of 0.5 mg/kg body weight per day is effective in most patients, but occasionally 1 mg/kg may be required.

Ivermectin. Single dose of 12 mg po in case of massive demodex infestation.

Rhinophyma and Telangiectasia. Treated by surgery or laser surgery with excellent cosmetic results. Web site *http://www.aad.org/pamphlets/rosacea.html.* The β-blocker carvedilol 6.5 mg bid has been reported to reduce erythema and telangiectasia.

Systemic. Oral antibiotics are more effective than topical treatment.

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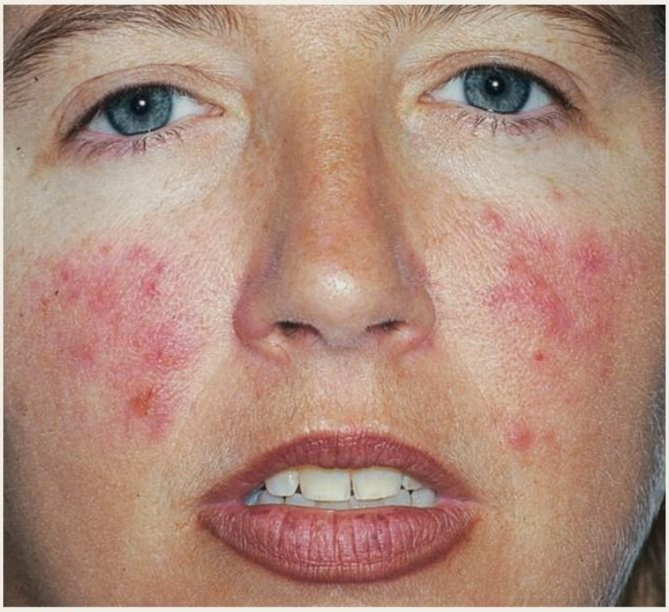


Figure 1-8. Rosacea Moderately severe rosacea in a 29-year-old female with persistent erythema, telangiectasia, red papules (stage II), and tiny pustules.

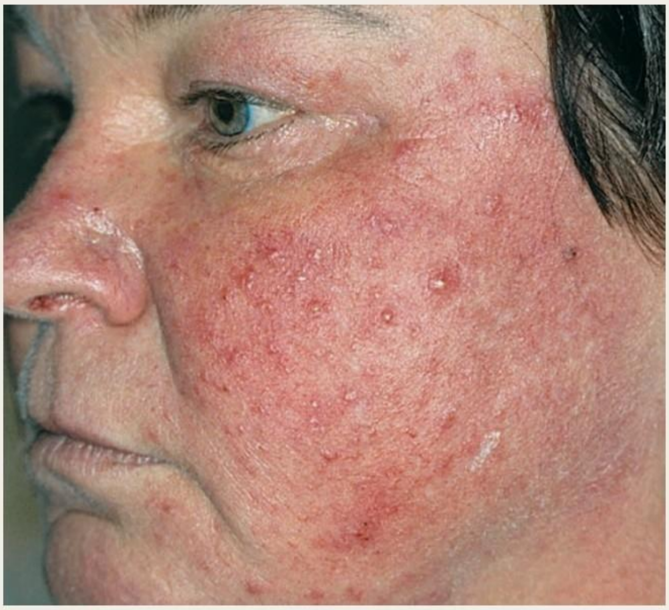
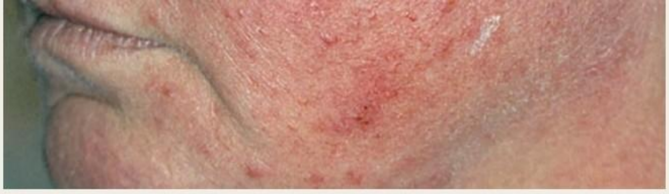


Figure 1-9. Rosacea, stages II–III Telangiectasia, papules and pustules, and some swelling in a 50-year-old woman. There are no comedones.



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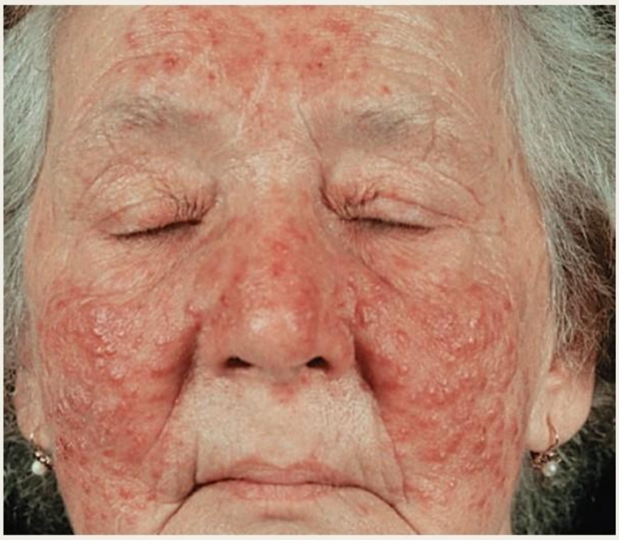


Figure 1-10. Papulopustular rosacea (early stage III) In this 65-year-old female, rosacea involves almost the entire face, sparing only the upper lip and chin. Papules and pustules have coalesced—again no comedones—and have already led to some swelling of the cheeks, which present "solid" edema.

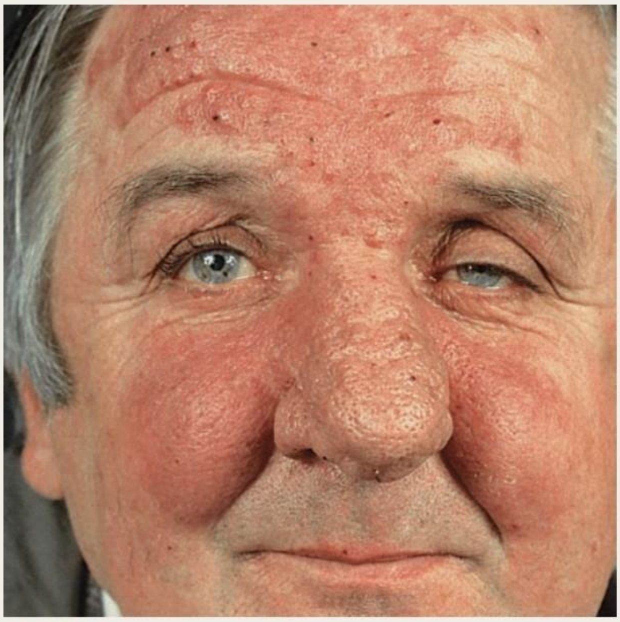


Figure 1-11. Rosacea (stage III) Here the persistent "solid" edema of the nose, forehead, and parts of the cheeks is the leading symptom. Papules, pustules, and crusted pustules are superimposed on this persistent edema. The enlarged nose feels rubbery and already represents rhinophyma.

Perioral Dermatitis ICD-9: 695.3 ◦ ICD-10: L71.0 □ ● → ◐*

Laboratory Examinations

Culture. Rule out *S. aureus* infection.

clear, then 500 mg daily for 1 month, then 250 mg daily for an additional month.

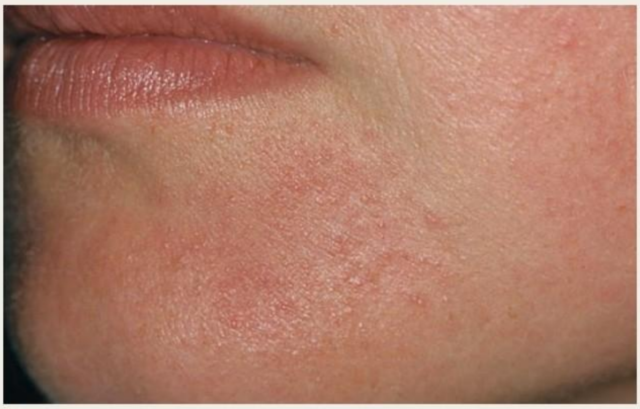


Figure 1-12. Perioral dermatitis Moderate involvement with early confluence of tiny papules and a few pustules in a perioral distribution in a young woman. Note typical sparing of the vermilion border (mucocutaneous junction).

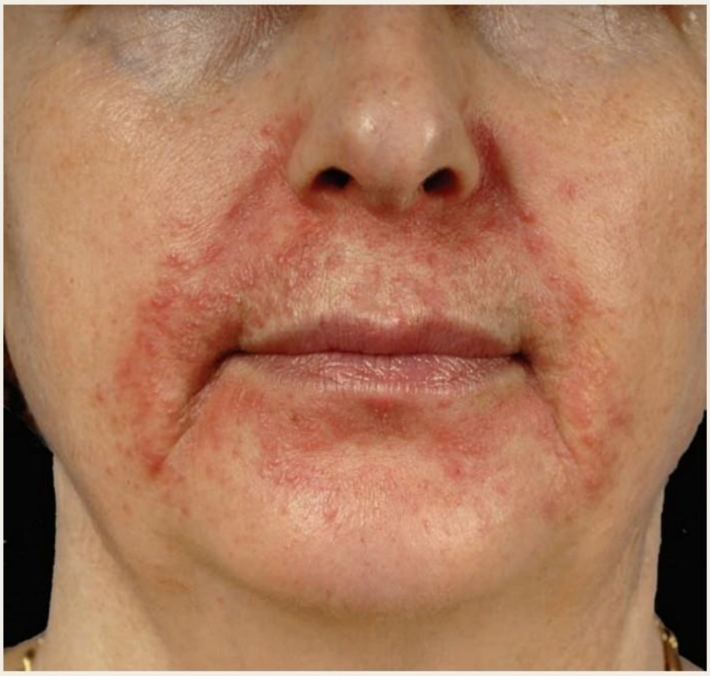


Figure 1-13. Perioral dermatitis Preferential location around the mouth and nasolabial folds and cheeks. This 38-year-old woman has been treated with fluorinated corticosteroids that led to a worsening of the condition.

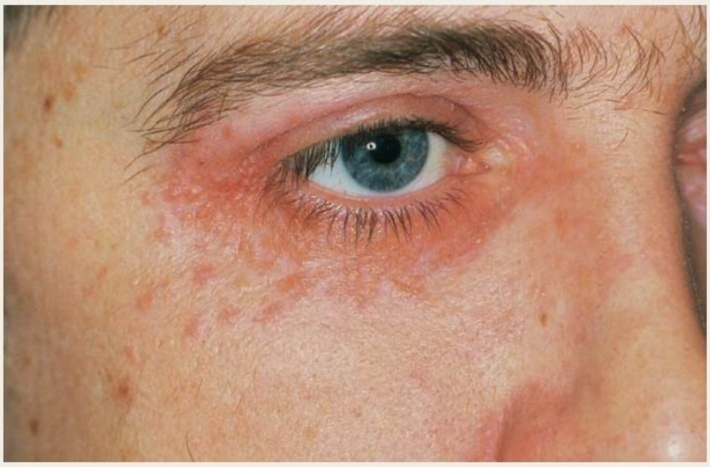


Figure 1-14. Periorbital dermatitis Note presence of tiny papules and a few pustules around the eye. This is a much less common site than the lesions around the mouth.

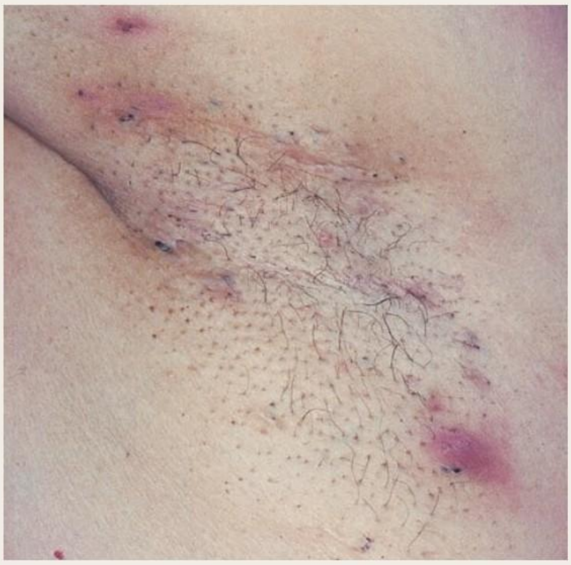


Figure 1-15. Hidradenitis suppurativa Many black comedones, some of which are paired, are a characteristic finding, associated with deep, exquisitely painful abscesses and old scars in the axilla.

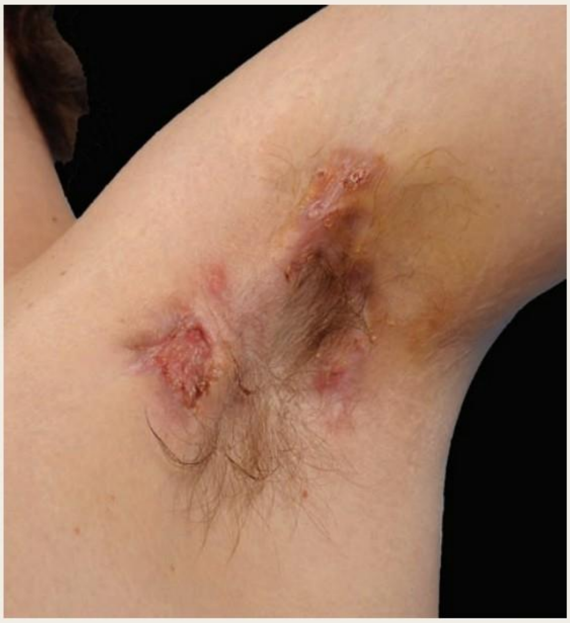


Figure 1-16. Hidradenitis suppurativa Multiple bulging and depressed scars, draining sinuses and larger ulcer in the axilla of a 24-year-old male.

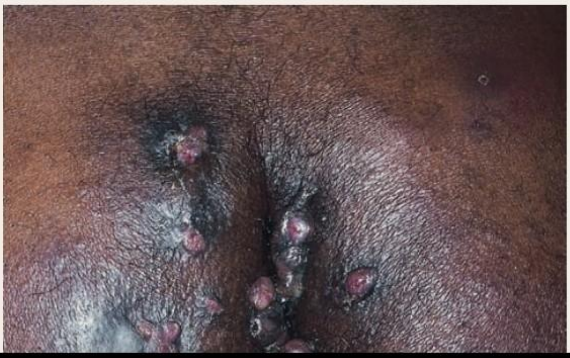
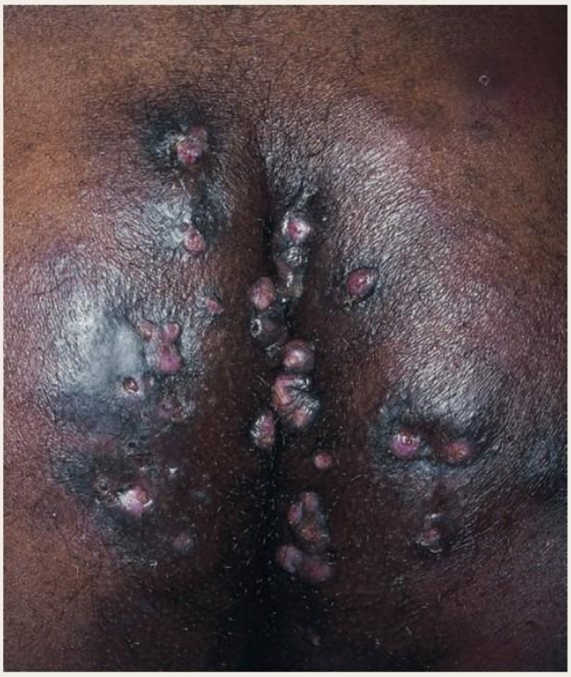


Figure 1-17. Hidradenitis suppurativa Severe scarring on the buttocks, inflammatory painful nodules with fistulas, and draining sinuses. When the patient sits down, pus will squirt from the sinus openings.



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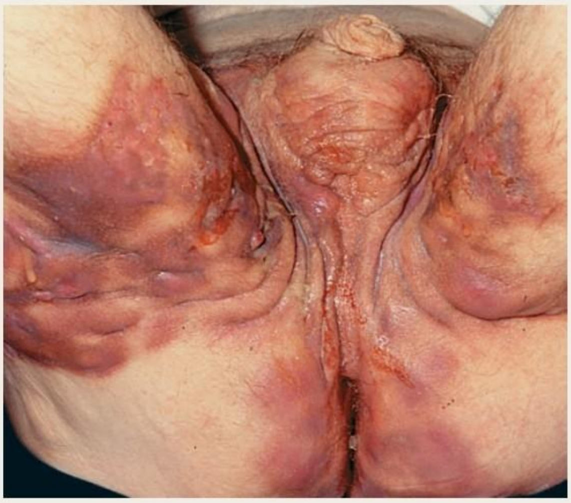


Figure 1-18. Hidradenitis suppurativa The entire perigenital and perianal skin as well as the buttocks and inner aspects of the thighs are involved in this 50-year-old male. There is considerable inflammation, and pressure releases purulent exudate from multiple sinuses. The patient had to wear a large diaper, because whenever he was seated, secretions would squirt from the sinuses.

Medical Management

Acute Painful *Nodule* and Abscess. Intralesional triamcinolone (3–5 mg/mL) into the wall followed by incision and drainage of abscess.
Chronic Low-Grade Disease. Oral antibiotics: erythromycin (250–500 mg qid), tetracycline (250–500 mg qid), or minocycline (100 mg bid);

Surgical Management

- Incise and drain acute abscesses.
- Excise chronic recurrent, fibrotic nodules, or sinus tracts.
- With extensive, chronic disease, complete excision of axilla or involved anogenital area extending down to fascia, requires split skin

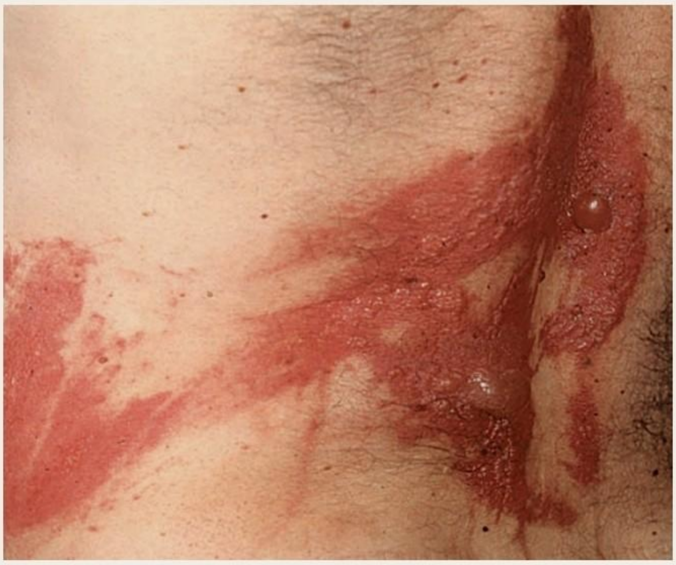


Figure 2-1. Acute irritant contact dermatitis following application of a cream containing nonylvanillamid and nicotinic acid butoxyethyl ester prescribed for lower back pain The "streaky pattern" indicates an outside job. The eruption is characterized by a massive erythema with vesiculation and blister formation and is confined to the sites exposed to the toxic agent.

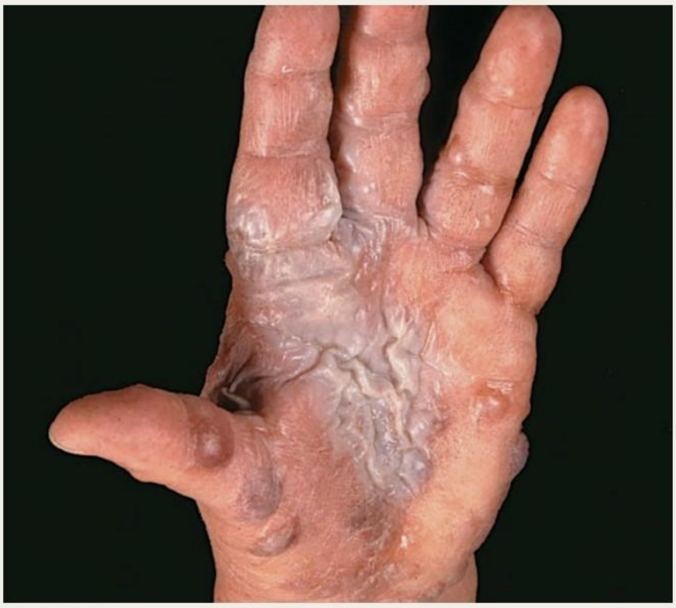


Figure 2-2. Acute irritant contact dermatitis on the hand due to an industrial solvent There is massive blistering on the palm.

Constitutional Symptoms

Usually none, but in widespread acute ICD

Clinical Manifestation

Symptoms. Stinging, smarting, burning, and

DERMATITIS

Cumulative ICD. Most common; develops slowly after repeated additive exposure to mild

gives way to ill-defined borders, lichenification.

Distribution. Usually on hands (Figs. 2-3 and 2-4). Usually starting at finger web spaces, spreading to sides and dorsal surface of hands,

Cumulative ICD. Most common; develops slowly after repeated additive exposure to mild irritants (water, soap, detergents, etc.), usually on hands. Repeated exposures to toxic or subtoxic concentrations of offending agents → disturbance of the barrier function that allows even subtoxic concentrations of offending agent to penetrate into the skin and elicit a chronic inflammatory response. Injury (e.g., repeated rubbing of the skin), prolonged soaking in water, or chronic contact after repeated, cumulative physical trauma—friction, pressure, and abrasions in individuals engaged in manual work (*traumatic ICD*).

Distribution. Usually on hands (Figs. 2-3 and 2-4). Usually starting at finger web spaces, spreading to sides and dorsal surface of hands, and then to palms. In housewives often starting on fingertips (*pulpitis*) (Fig. 2-3). Rarely in other locations exposed to irritants and/or trauma, e.g., in violinists on mandible or neck, or on exposed sites as in *airborne ICD* (see below).

Duration. Chronic, months to years.

Constitutional Symptoms

None, except when infection occurs. Chronic ICD (e.g., hand dermatitis; see below) can become a severe occupational and emotional problem.

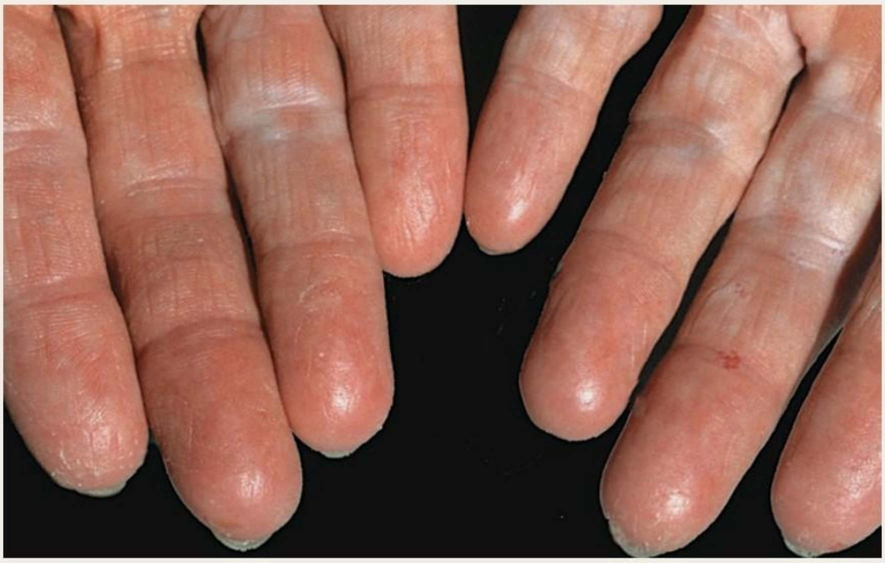
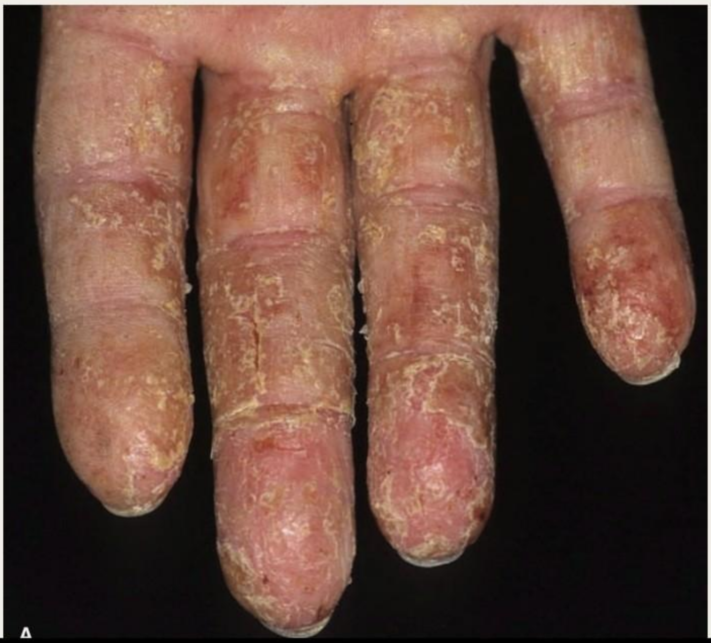


Figure 2-3. Early chronic irritant contact dermatitis in a housewife This has resulted from repeated exposure to soaps and detergents. Note glistening fingertips (pulpitis).



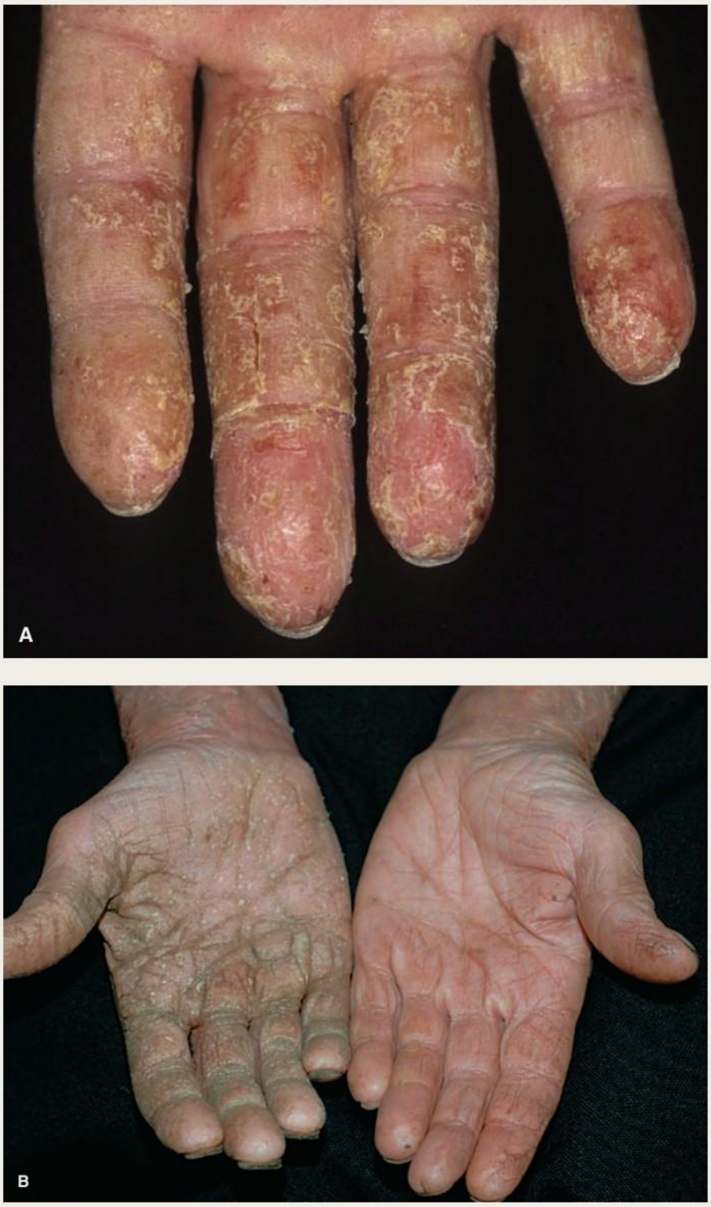


Figure 2-4. (A) Chronic irritant dermatitis with acute exacerbation in a housewife The patient used turpentine to clean her hands after painting. Erythema, fissuring, and scaling. Differential diagnosis is allergic contact dermatitis and palmar psoriasis. Patch tests to turpentine were negative. **(B) Irritant contact dermatitis in a construction worker who works with cement** Note the hyperkeratoses, scaling, and fissuring. There is also minimal pustulation. Note that right (dominant working) hand is more severely affected than left hand.

Laboratory Examination

Histopathology. In acute ICD, epidermal cell necrosis, neutrophils, vesiculation, and necrosis. In chronic ICD, acanthosis, hyperkeratosis, and lymphocytic infiltrate.

Patch Tests. These are negative in ICD unless ACD is also present (see below).

cases, 6 weeks or longer may be required. In the setting of occupational ICD, only one-third of individuals have complete remission and two-thirds may require allocation to another job; atopic individuals have a worse prognosis. In cases of chronic subcritical levels of irritant, some workers develop tolerance, or "hardening."

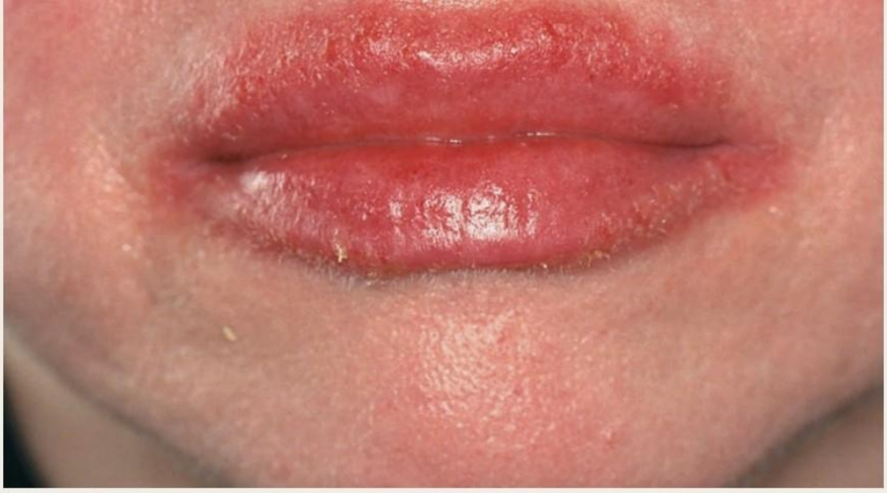


Figure 2-5. Acute allergic contact dermatitis on the lips due to lipstick The patient was hypersensitive to eosin. Note bright erythema, microvesiculation. At close inspection, a papular component can be discerned. At this stage, there is still sharp margination.

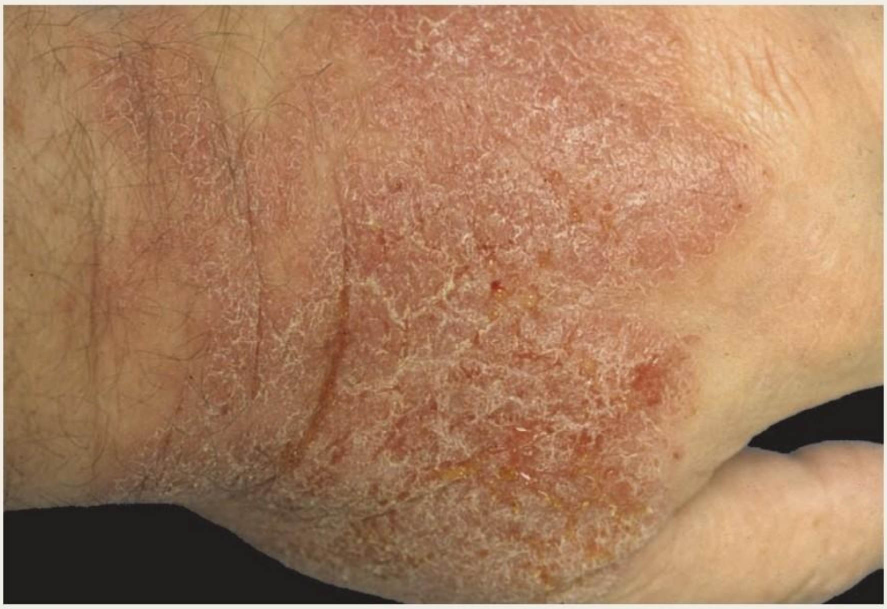


Figure 2-6. Allergic contact dermatitis of hands: chromates Confluent papules, vesicles, erosions, and crusts on the dorsum of the left hand in a construction worker who was allergic to chromates.

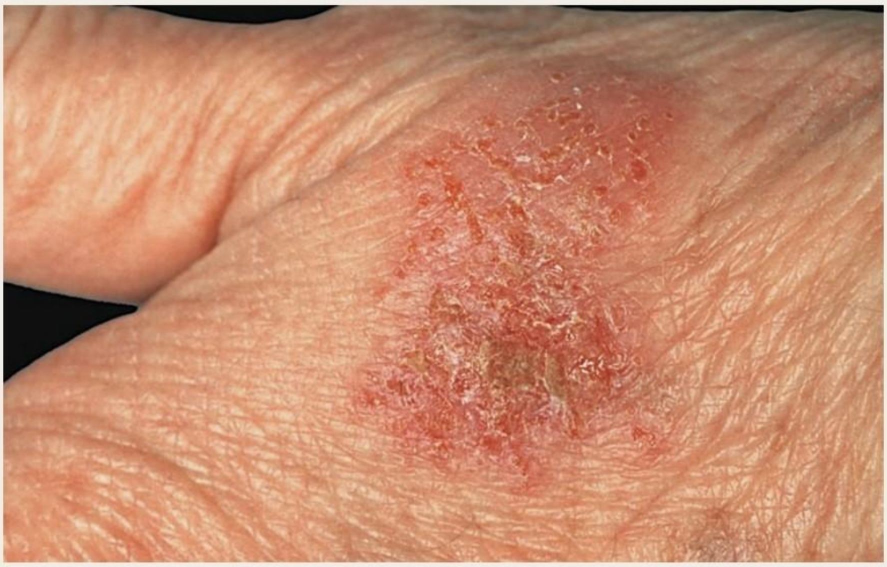


Figure 2-7. Allergic contact dermatitis due to nickel, subacute Note a mix of papular, vesicular, and crusted lesions and loss of sharp margination. The patient was a retired watchmaker who used a metal clasp on the dorsum of the left hand while repairing watches. He was known to be allergic to nickel.

turns to a black resin on exposure to air. Cashew oil, unroasted cashew nuts (heat destroys

Distribution. Most commonly on exposed extremities, where contact with the plant

occurs; blotting can transfer to any exposed site; palms/soles usually spared; however, lateral fingers can be involved.

Clothing-Protected Sites. Oleoresin can penetrate damp clothing onto covered skin; wearing clothing previously contaminated with resin can reexpose the skin.

Nonexposed Sites. "Id"-like reaction or some systemic absorption can be associated with disseminated urticarial, erythema multiforme-like, or scarlatiniform lesions away from sites of exposure in some individuals with well-established APD.

Laboratory Examinations

Dermatopathology. See ACD, above.

Patch Tests with Pentadecylcatechols. *Contraindicated* as it can sensitize individual to hapten.

Diagnosis

By history and clinical findings only.

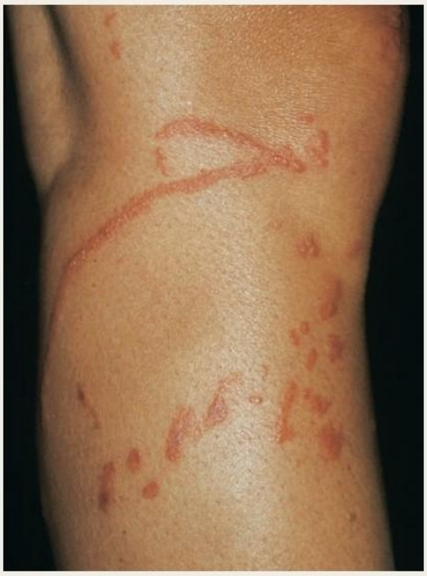


Figure 2-8. Allergic phytodermatitis of leg: poison ivy Linear vesicular lesions with erythema and edema on the calf at sites of direct contact of the skin 5 days after exposure with the poison ivy leaf.

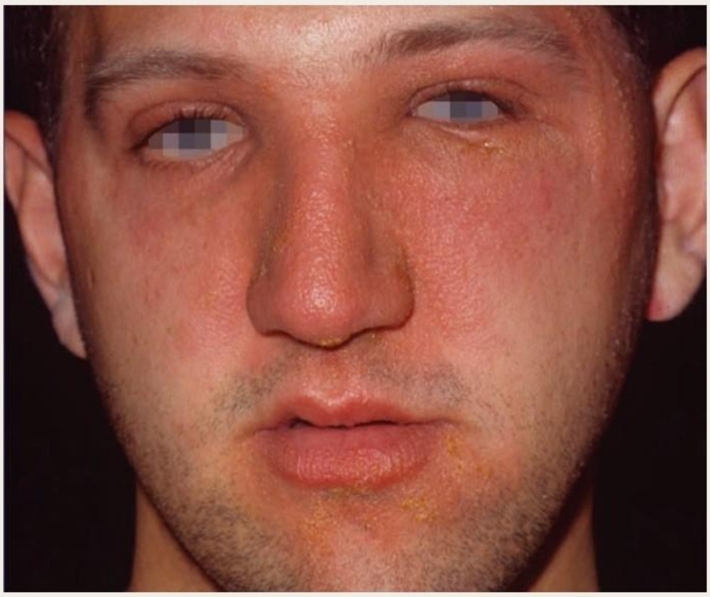
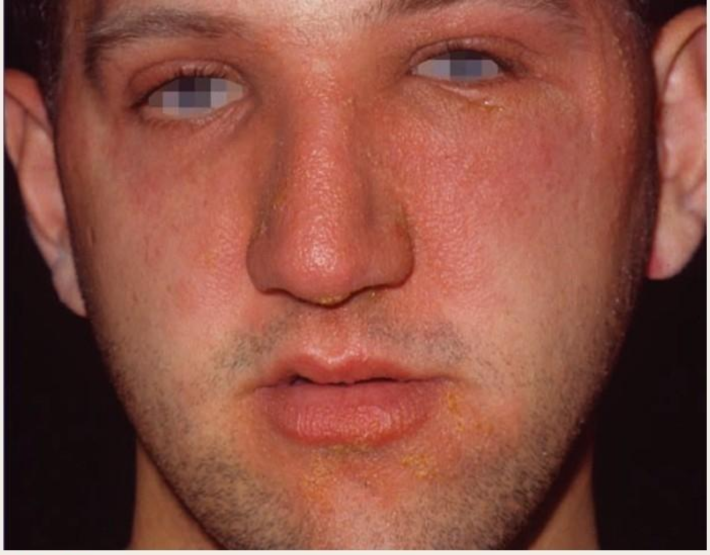


Figure 2-9. Allergic phytodermatitis of the face: poison ivy Extremely pruritic, erythema, edema, and microvesiculation in the periorbital and perioral area in a previously sensitized young man, occurring 3 days after exposure.



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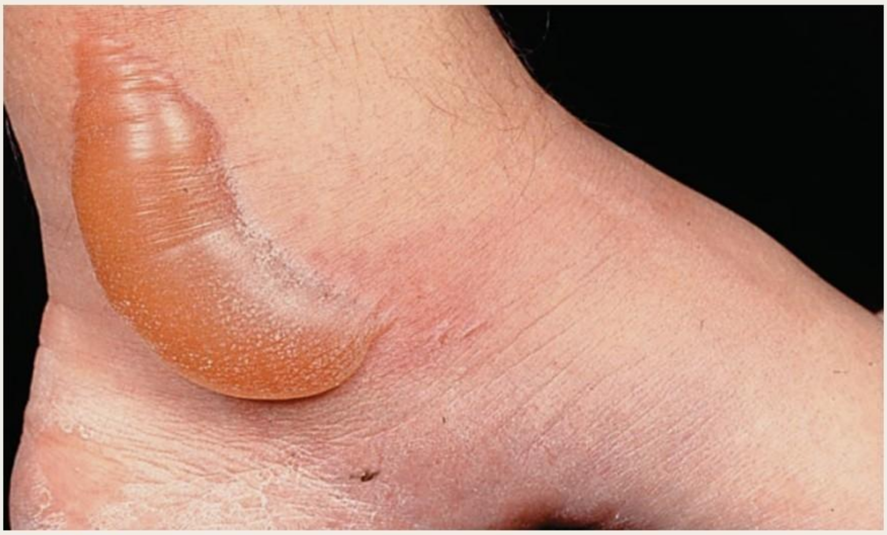


Figure 2-10. Acute allergic phytodermatitis, bullous This eruption occurred in a patient who had walked barefoot through a forest. It later spread as a papular eruption to the rest of the body. Similar lesions were present on the other foot and lower leg. Differential diagnosis included acute bullous contact dermatitis to caterpillars. Phytophotodermatitis was excluded because at the time of exposure, there was a heavily clouded sky and a papular eruption occurred later on. Caterpillar dermatitis was excluded because of the multiplicity of the lesions and because upon patch testing, the patient was positive to *Toxicodendron* haptens. Note, patch testing to urushiol is no longer done to avoid sensitization of patients.

Differential Diagnosis

ACD to other allergens, phytophotodermatitis (see Section 10), soft-tissue infection (cellulitis, erysipelas), AD, inflammatory dermatophytosis, early herpes zoster, and fixed drug eruption.

Other Special Forms of ACD

Systemic ACD

- After systemic exposure to an allergen to which the individual had prior ACD
- A delayed T-cell–mediated reaction
- Examples: ACD to ethylenediamine → subsequent reaction to aminophylline (which contains ethylene diamine); poison ivy dermatitis → subsequent reaction to ingestion of cashew nuts; also

Airborne ACD

- Contact with airborne allergens in exposed body sites, notably the face (Fig. 2-11); also including eyelids, "V" of the neck, arms, and legs
- In contrast to airborne ICD, papular from the beginning, extremely itchy
- Prolnged repetitive exposure leads to dry, lichenified ACD with erosions and crusting (Fig. 2-11)
- Due to plant allergens, especially from compositae, natural resins, woods, and essential oils volatizing from aroma therapy

Management of ACD

Termination of Exposure. Identify and remove (see Section 10), soft-tissue infection (cellulitis, erysipelas), AD, inflammatory dermatophytosis, early herpes zoster, and fixed drug eruption.

Other Special Forms of ACD

Systemic ACD

- After systemic exposure to an allergen to which the individual had prior ACD
- A delayed T-cell–mediated reaction
- Examples: ACD to ethylenediamine → subsequent reaction to aminophylline (which contains ethylene diamine); poison ivy dermatitis → subsequent reaction to ingestion of cashew nuts; also antibiotics, sulfonamides, propylene glycol, metal ions, sorbic acid, fragrances

- Contact with airborne allergens in exposed body sites, notably the face (Fig. 2-11); also including eyelids, "V" of the neck, arms, and legs
- In contrast to airborne ICD, papular from the beginning, extremely itchy
- Prolnged repetitive exposure leads to dry, lichenified ACD with erosions and crusting (Fig. 2-11)
- Due to plant allergens, especially from compositae, natural resins, woods, and essential oils volatizing from aroma therapy

Management of ACD

Termination of Exposure. Identify and remove the etiologic agent.

Topical Therapy. Topical glucocorticoid ointments/gels (classes I–III). Larger vesicles may be drained, but tops should not be removed. Wet dressings with cloths soaked in Burow's solution changed every 2–3 h. Airborne ACD may require systemic treatment. Pimecrolimus and tacrolimus are effective in ACD but to a lesser degree than glucocorticoids.

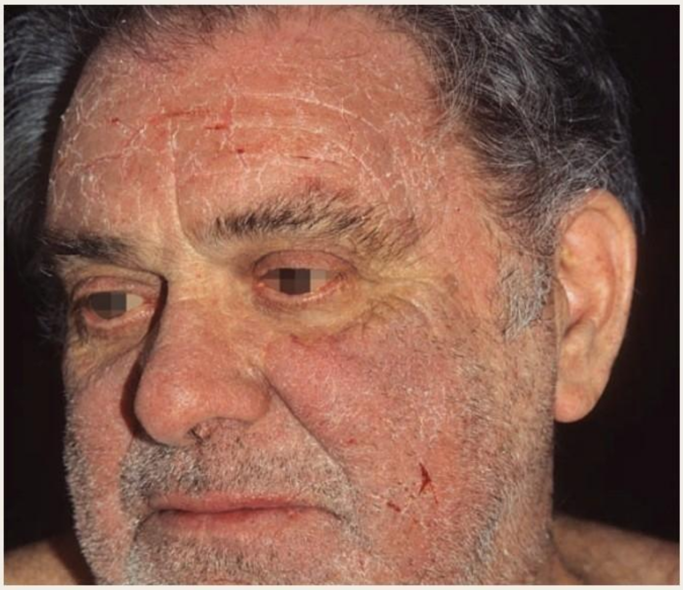


Figure 2-11. Airborne allergic contact dermatitis of the face Extremely itchy, confluent, papular, erosive, and crusted/scaly lesions with lichenification on the forehead, nose, and cheeks following exposure to pinewood dust.

Systemic Therapy. Glucocorticoids are indicated if severe and in airborne ACD. Prednisone beginning at 70 mg (adults), tapering by 5–10 mg/d over a 1- to 2-week period. In airborne ACD where complete avoidance of allergen may be impossible, immunosuppression with oral cyclosporine may become necessary.

Atopic Dermatitis ICD-9: 691.8 ∘ ICD-10: L20 ☐ ◐

- An acute, subacute, or chronic relapsing skin disorder.
- Very common in infancy.
- Prevalence peak of 15–20% in early childhood.
- Characterized principally by dry skin and pruritus; consequent rubbing leads to increased inflammation and lichenification and to further itching and scratching: *itch–scratch cycle.*
- Diagnosis is based on clinical findings.
- Often associated with a personal or family history of AD, allergic rhinitis, and asthma; 35% of infants with AD develop asthma later in life.
- Associated with skin barrier dysfunction, IgE reactivity.
- Genetic basis influenced by environmental factors; alterations in immunologic responses in T cells, antigen processing, inflammatory cytokine release, allergen sensitivity, infection.

Synonyms: IgE dermatitis, "eczema," atopic eczema.

Epidemiology

Age of Onset. First 2 months of life and by the first years in 60% of patients; 30% by age 5, and only 10% between age 6 and 20 years. Rarely AD has an adult onset.

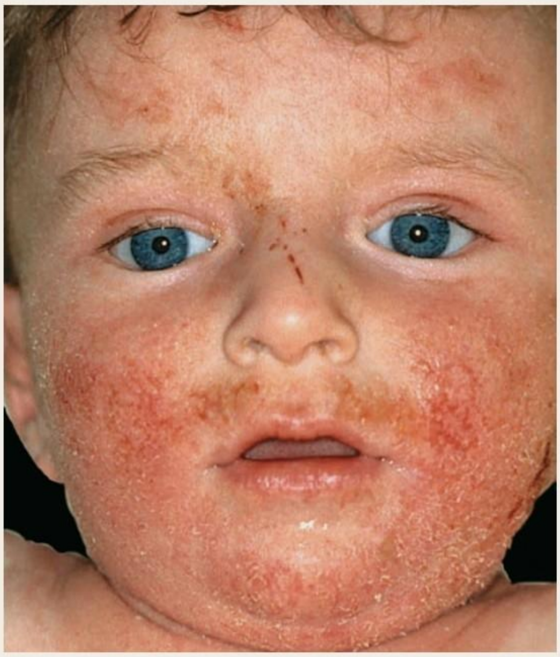
Gender. Slightly more common in males than in females.

sitized by the interaction of the antigen with IgE (reaginic or skin-sensitizing antibody). Epidermal Langerhans cells possess high-affinity IgE receptors through which an eczema-like reaction can be mediated. Acute inflammation in AD is associated with a predominance of interleukin (IL) 4 and IL-13 expression, and chronic inflammation in AD with increased IL-5, granulocyte-macrophage colony-stimulating factor, IL-12, and interferon-γ. Thus, skin

Childhood-Type AD. The lesions are papular, lichenified plaques, erosions, crusts, especially on the antecubital and popliteal fossae (Figs. 2-15A and B), the neck and face; may be generalized.

Adult-Type AD. There is a similar distribution, mostly flexural but also face and neck, with lichenification and excoriations being the most conspicuous symptoms (Fig. 2-17B). May be generalized.

Figure 2-12. Atopic dermatitis: infantile Puffy face, confluent erythema, papules, microvesiculation, scaling, and crusting.



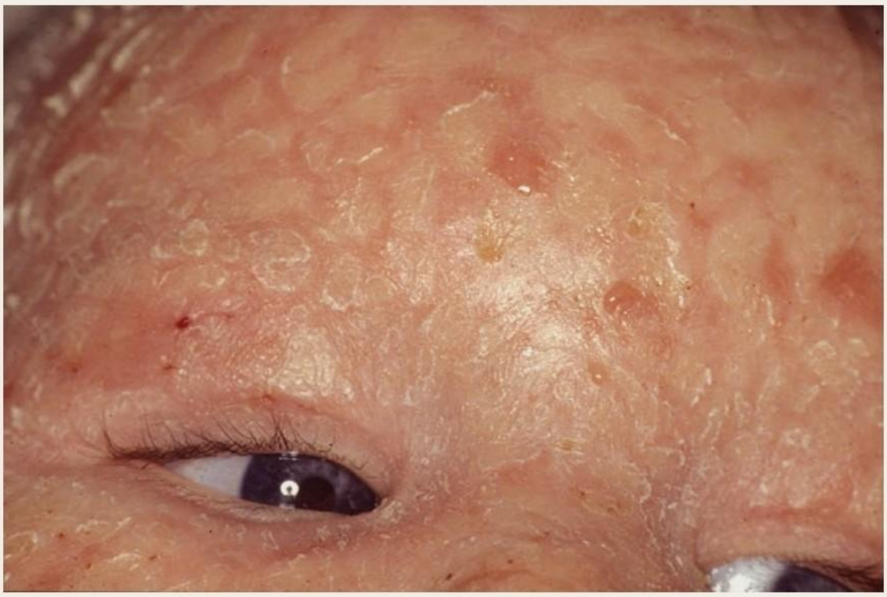


Figure 2-13. Atopic dermatitis: infantile type Skin of forehead is dry, cracked, and scaly. In addition, there are oozing erosions.

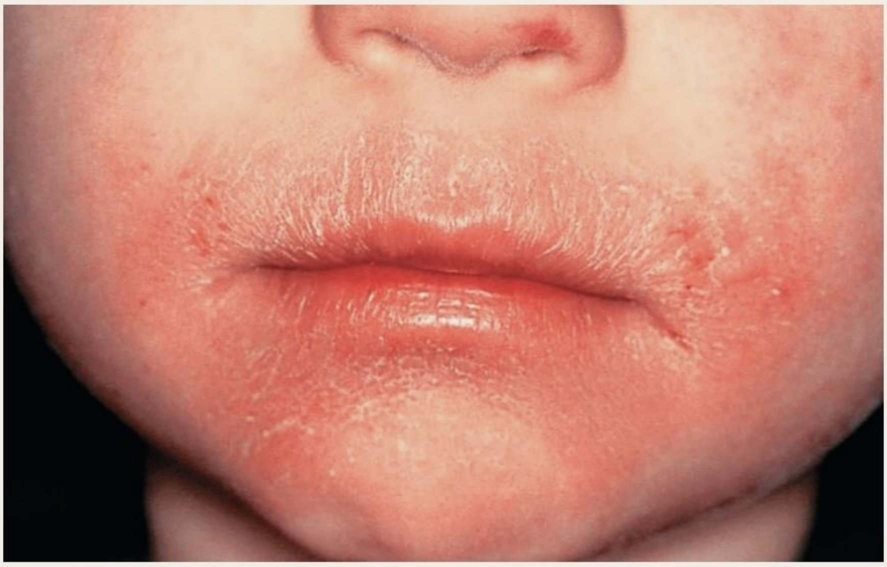


Figure 2-14. Childhood atopic dermatitis A typical localization of atopic dermatitis in children is the region around the mouth. In this child, there is lichenification and fissuring and crusting.

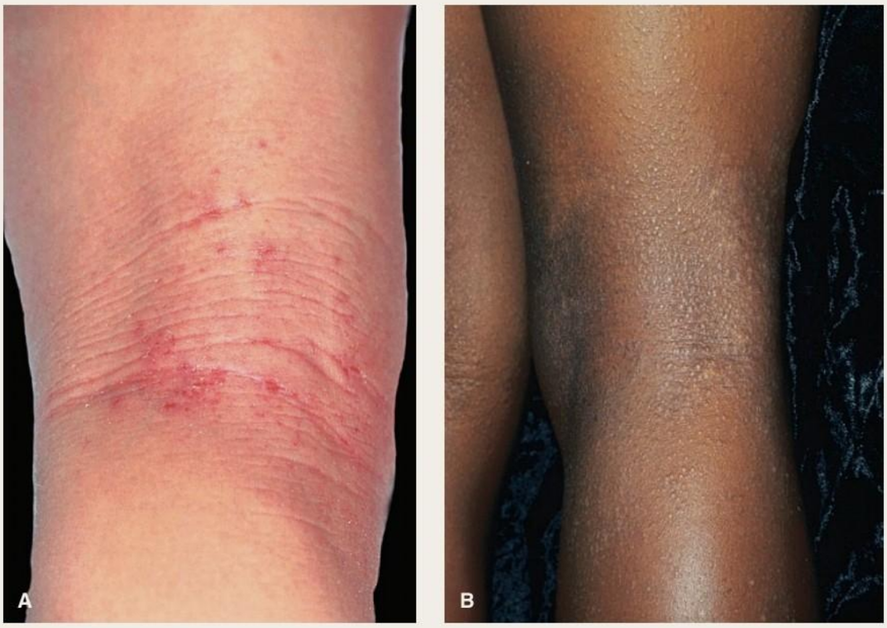
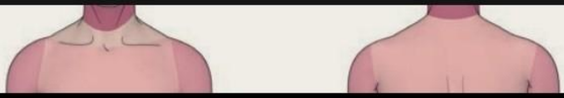


Figure 2-15. (A) Childhood atopic dermatitis One of the hallmarks of atopic dermatitis is lichenification in the flexural regions as shown in this picture. Note the thickening of the skin with exaggerated skin lines and erosions. **(B)** Atopic dermatitis in black child. Pruritic follicular papules on posterior leg. Follicular eczema pattern is more common in African and Asian children.



"White" Dermatographism. Stroking of involved skin will not lead to redness as in normal skin but to blanching; delayed blanch to cholinergic agents. *Ichthyosis vulgaris* and *keratosis pilaris* (see Section 4) occur in 10% of patients. *Vernal conjunctivitis* with papillary hypertrophy or cobblestoning of upper eyelid conjunctiva. Rare *atopic keratoconjunctivitis* is disabling, may result in corneal scarring. *Keratoconus* is rare. *Cataracts* occur in a small percentage.

glucagonoma syndrome, histidinemia, phenylketonuria; also, some immunologic disorders including Wiskott–Aldrich syndrome, X-linked agammaglobulinemia, hyper-IgE syndrome, and selective IgA deficiency; Langerhans cell histiocytosis, Letterer–Siwe type.

Laboratory Examinations

Bacterial Culture. Colonization with *S. aureus* is very common in the nares and in the involved

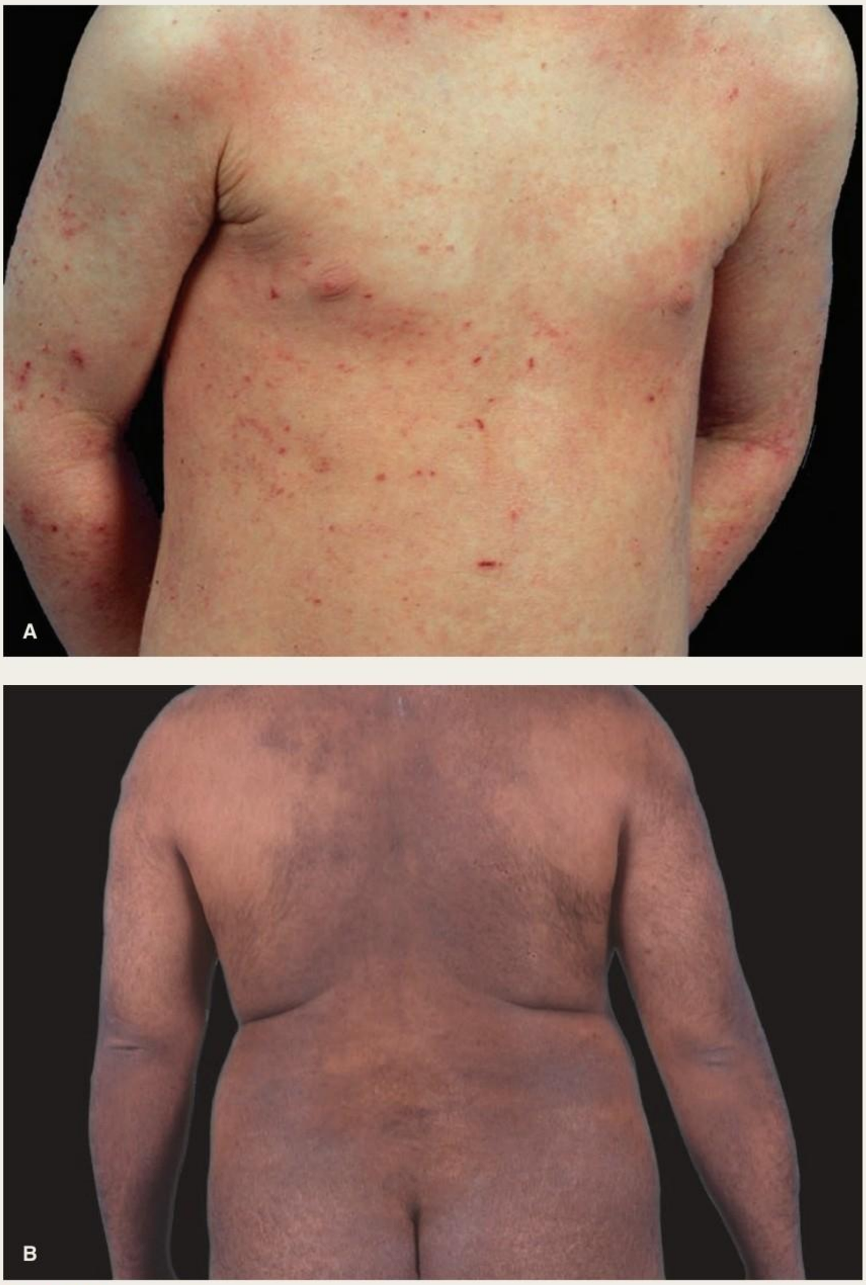


Figure 2-17. (A) Childhood atopic dermatitis This is a generalized eruption consisting of confluent, inflammatory papules that are erosive, excoriated, and crusted. **(B)** Generalized eruption of follicular papules that are more heavily pigmented than normal skin in a 53-year-old woman of African extraction. There is extensive lichenification.

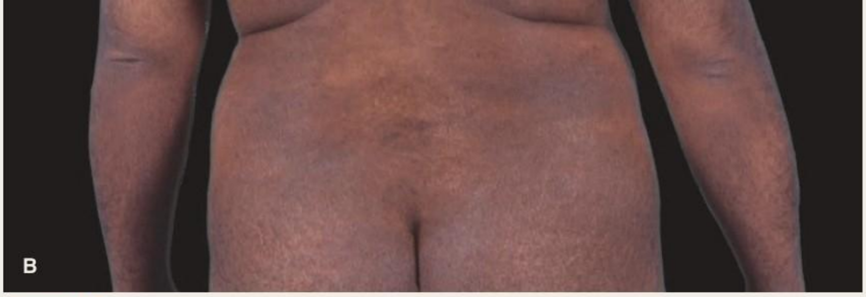


Figure 2-17. (A) Childhood atopic dermatitis This is a generalized eruption consisting of confluent, inflammatory papules that are erosive, excoriated, and crusted. **(B)** Generalized eruption of follicular papules that are more heavily pigmented than normal skin in a 53-year-old woman of African extraction. There is extensive lichenification.

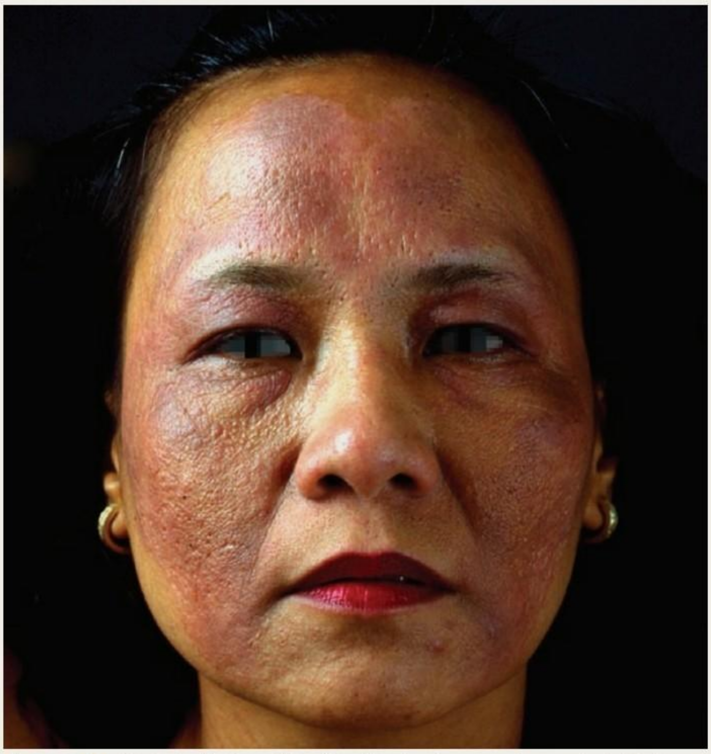


Figure 2-18. Adult atopic dermatitis Lichenification does not only occur in the big flexural folds but may also affect the face as in this 53-year-old woman of Indonesian extraction.

skin; almost 90% of patients with severe AD are secondarily colonized/infected. Look out for methicillin-resistant *S. aureus* (MRSA).

Viral Culture. Rule out herpes simplex virus (HSV) infection in crusted lesions (eczema herpeticum; see Section 27).

Blood Studies. Increased IgE in serum, eosinophilia. HSV antigen detection for diagnosis of acute HSV infection.

Dermatopathology. Various degrees of acanthosis with rare intraepidermal intercellular edema (spongiosis). The dermal infiltrate is composed of lymphocytes, monocytes, and mast cells with few or no eosinophils.

Special Forms of AD

Hand Dermatitis. Aggravated by wetting and washing with detergents, harsh soaps, and *disinfectants;* leads to ICD in the atopic. Clinically indistinguishable from “normal” ICD (see page 21).

Exfoliative Dermatitis (See Section 8). Erythroderma in patients with extensive skin involvement. Generalized redness, scaling, weeping, crusting, lymphadenopathy, fever, and systemic toxicity.

Complications

Secondary infection with *S. aureus* and HSV (eczema herpeticum, see Section 28). Rarely keratoconus, cataracts, and keratoconjunctivitis with secondary herpetic infection and corneal ulcers.

Course and Prognosis

Untreated involved sites persist for months or years. Spontaneous, more or less complete remission during childhood occurs in >40% with occasional, more severe recurrences during adolescence. In many patients, the disease persists for 15–20 years, but is less severe. Thirty to fifty area, scrotum, and groin.

In black skin, lichenification may assume a special type of pattern consisting of multiple small (2–3 mm) closely set papules, a "follicular" pattern (as in Fig. 2-15B).

Intralesional triamcinolone is often highly effective in smaller lesions (3 mg/mL; higher concentrations may cause atrophy). Oral hydroxyzine, 25–50 g at night, may be helpful.

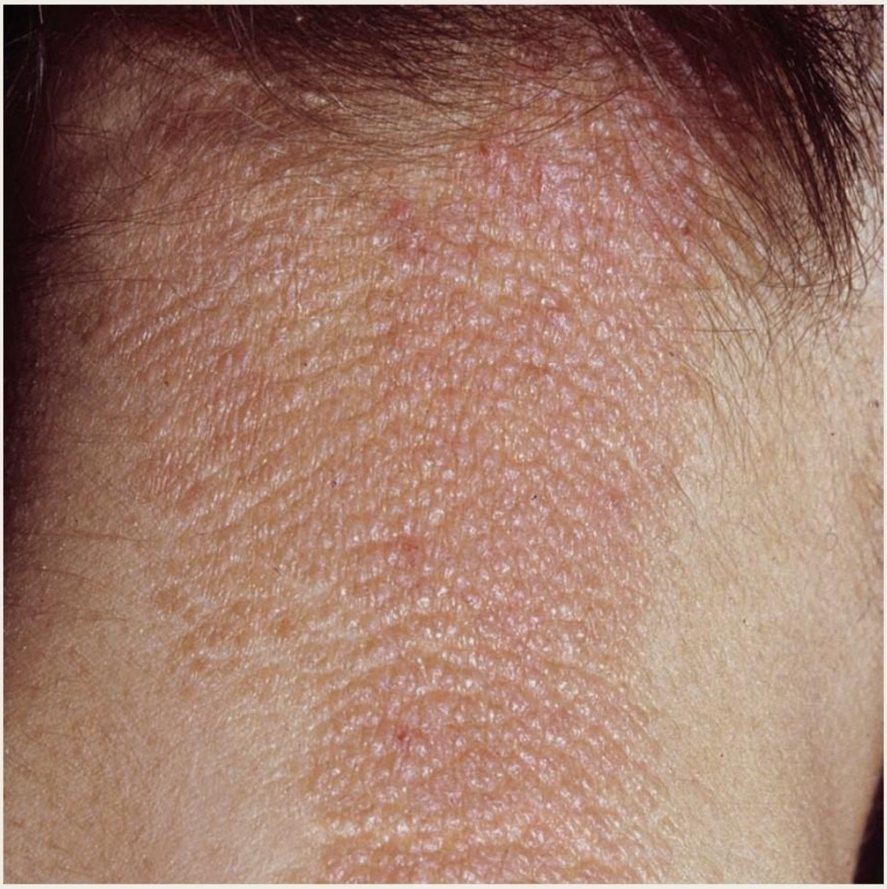


Figure 2-19. Lichen simplex chronicus Confluent, papular, follicular eczema, creating a plaque of lichen simplex chronicus of the posterior neck and occipital scalp. Condition had been present for many years as a result of chronic rubbing of the area.

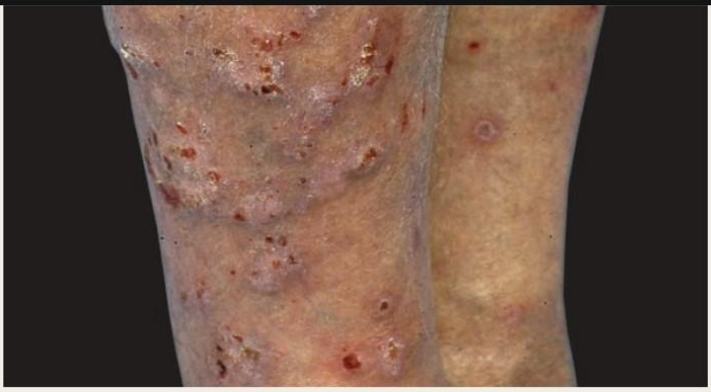


Figure 2-20. Prurigo nodularis Multiple, firm, excoriated nodules arising at sites of chronically picked or excoriated skin. Often occurring in patients with atopy but also without it. In this 56-year-old patient, the extreme pruritus necessitated multiple hospitalizations.

Dyshidrotic Eczematous Dermatitis ICD-9: 705.81 ∘ ICD-10: L30.1 ◧ ◐

- Dyshidrotic eczema is a special vesicular type of hand and foot dermatitis.
- An acute, chronic, or recurrent dermatosis of the fingers, palms, and soles.
- Sudden onset of many deep-seated pruritic, clear "tapioca-like" vesicles (Fig. 2-21).
- Large bullae can occur (pompholyx).
- Later scaling fissures and lichenification.
- Itching and when erosions are present pain.
- Secondary bacterial infection: pustules, cellulitis, lymphangitis, and painful lymphadenopathy.
- Recurrent attacks are the rule.
- Treatment: topical high-potency corticosteroids, intralesional triamcinolone 3 mg/mL for small areas; in severe cases, a short course of prednisone: starting with 70 mg and tapering by 10 or 5 mg over 7 or 14 days; systemic antibiotics for secondary infection and PUVA either oral or as "soaks" (see page 60).

Synonyms: Pompholyx, vesicular palmar eczema.

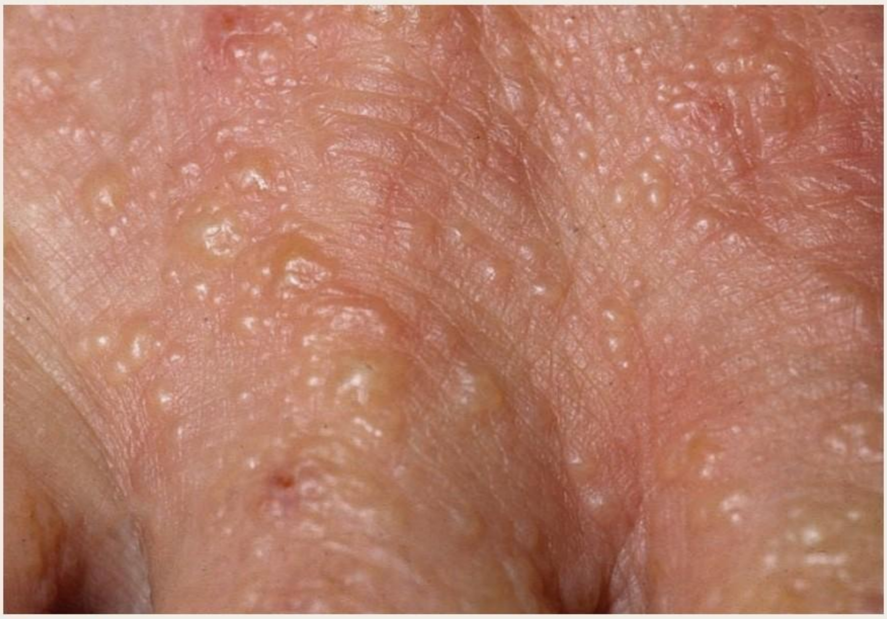


Figure 2-21. Dyshidrotic eczematous dermatitis Confluent tapioca-like vesicles and crusted (excoriated) erosions on the dorsum of fingers and finger webs.

Nummular Eczema ICD-9: 692.9 ◦ ICD-10: L30.9

- Nummular eczema is a chronic, pruritic, inflammatory dermatitis occurring in the form of coin-shaped plaques composed of grouped small papules and vesicles on an erythematous base (Fig. 2-22).
- It is especially common on the extremities during winter months when xerosis is maximal; often seen in atopic individuals.
- *S. aureus* is often present but pathogenic significance not proven.
- Very pruritic. Course is chronic, lesions last from weeks to months.
- Management: Hydrate skin with hydrated moisturizer or moisturizing cream, topical glucocorticosteroids or 2–5% crude coal tar ointment. PUVA or UVB-311 therapy very effective.

Synonyms: Discoid eczema, microbial eczema.

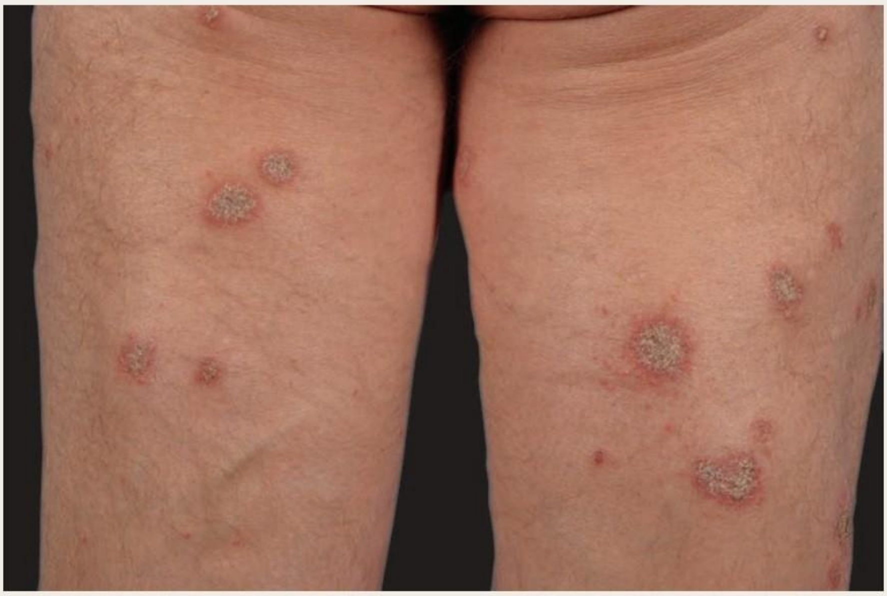


Figure 2-22. Nummular eczema Pruritic, round, nummular (coin-shaped) plaques with erythema, scales, and crusts on the posterior legs.

Autosensitization Dermatitis ICD-9: 692.9 ◦ ICD-10: L30.9

- An often unrecognized generalized pruritic dermatitis directly related to a primary dermatitis elsewhere.
- For example, a patient with venous stasis dermatitis on the lower legs may develop pruritic, symmetric, scattered, erythematous, maculopapular, or papulovesicular lesions on the trunk, forearms, thighs, or legs.
- These persist and spread until the basic underlying primary dermatitis is controlled.
- Similarly, autosensitization may occur as an "id" reaction in inflammatory tinea pedis and manifests as a dyshidrosiform, vesicular eruption on the feet and hands (Fig. 2-23) and papulovesicular eczematoid lesions on the trunk.
- The phenomenon results from the release of cytokines in the primary dermatitis, as a result of sensitization. These cytokines circulate in the blood and heighten the sensitivity of the distant skin areas.
- The diagnosis of autosensitization dermatitis is often *post hoc;* i.e., the distant eruption disappears when the primary dermatitis is controlled.
- Oral glucocorticoids hasten the disappearance of the lesions.

papules of varying size (5–20 mm), or patches, rather sharply marginated (Fig. 2-24). On the scalp, there is mostly marked scaling ("dandruff"),

infants, often associated with purpura), acrodermatitis enteropathica, zinc deficiency, pemphigus foliaceus, glucagonoma syndrome.

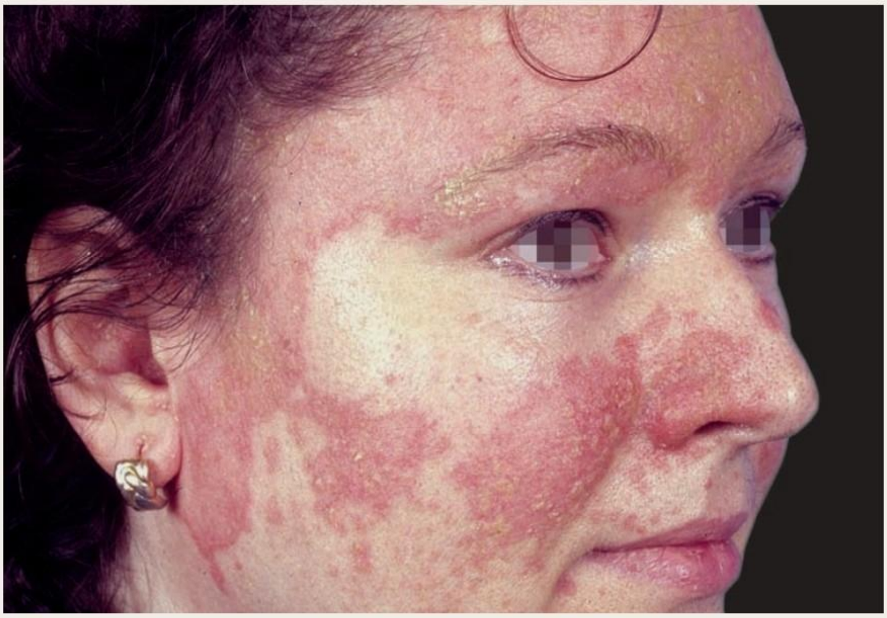


Figure 2-24. Seborrheic dermatitis of face: adult type Erythema and yellow-orange scaling the forehead, cheeks, nasolabial folds. Scalp and retroauricular areas were also involved.

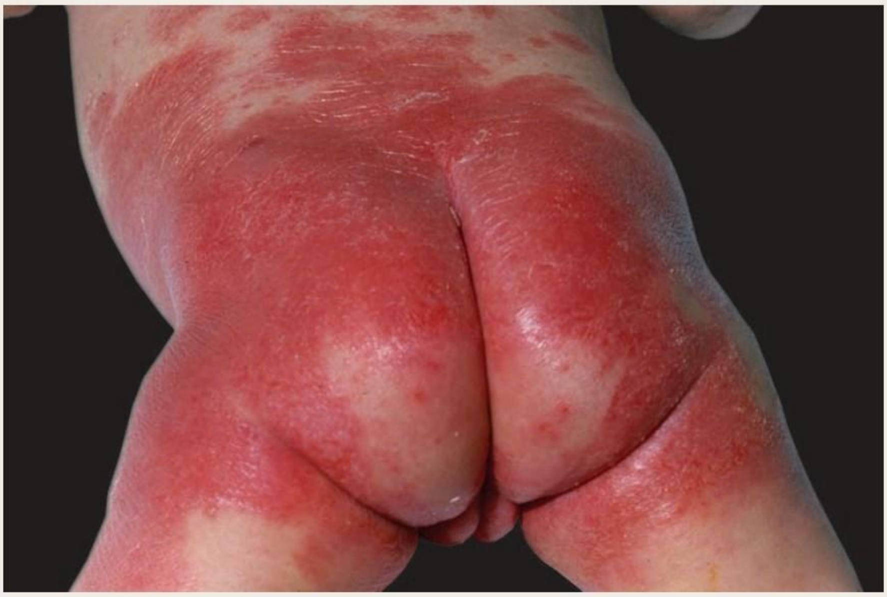


Figure 2-25. Seborrheic dermatitis: infantile type Erythema scales and crusting in the diaper region of an infant. This is difficult to distinguish in the diaper region from psoriasis and *Candida* has to be ruled out by KOH.

Laboratory Studies

Dermatopathology. Focal parakeratosis, with few neutrophils, moderate acanthosis, spongiosis (intercellular edema), and nonspecific inflammation of the dermis. A characteristic feature is neutrophils at the tips of the dilated follicular openings, which appear as crusts/

shampoo, 2% ketoconazole shampoo, and application of 1–2.5% hydrocortisone cream, 2% ketoconazole cream, and 1% pimecrolimus cream.

Face and Trunk. *Ketoconazole shampoo*, 2%. Glucocorticoid cream and lotions: initially 1% or 2.5% hydrocortisone cream; in more resistant

- slight scaling.
- The incessant pruritus can lead to lichenification, which can even persist when the environmental conditions have been corrected.
- The disorder results from too frequent bathing in hot soapy baths or showers and/or in older

skin is inflamed, use medium-potency glucocorticoid ointments, applied twice daily until the eczematous component has resolved.

Synonyms: Eczema *craquelé* (French *craquelé*, "marred with cracks," such as in old china and ceramic tile).

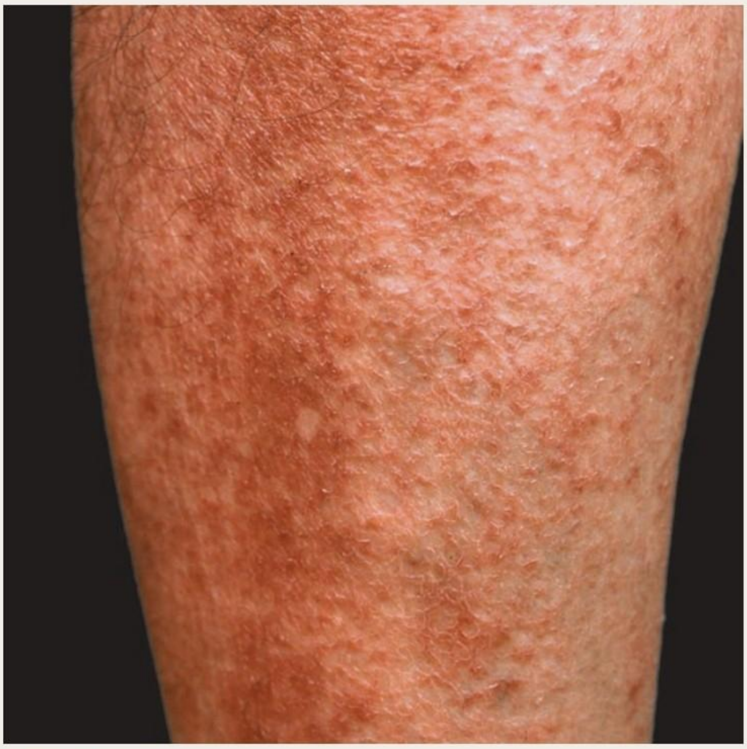


Figure 2-26. Asteatotic dermatitis In this 65-year-old man, lesions have coalesced to involve the entire skin of the lower leg.

SECTION 3

Psoriasis and Psoriasiform Dermatoses

Psoriasis

- Psoriasis affects 1.5–2% of the population in Western countries. Worldwide occurrence.
- A chronic disorder with polygenic predisposition and triggering environmental factors such as bacterial infection, trauma, or drugs.
- Several clinical expressions. Typical lesions are chronic, recurring, scaly papules, and plaques. Pustular eruptions and erythroderma occur.
- Clinical presentation varies among individuals, from those with only a few localized plaques to those with generalized skin involvement.
- Psoriatic erythroderma in psoriasis involving the entire skin.
- Psoriatic arthritis occurs in 10–25% of the patients.

Classification

Psoriasis vulgaris
  Acute guttate
  Chronic stable plaque
  Palmoplantar
  Inverse

Psoriatic erythroderma
Pustular psoriasis
  Pustular psoriasis of von Zumbusch
  Palmoplantar pustulosis
  Acrodermatitis continua

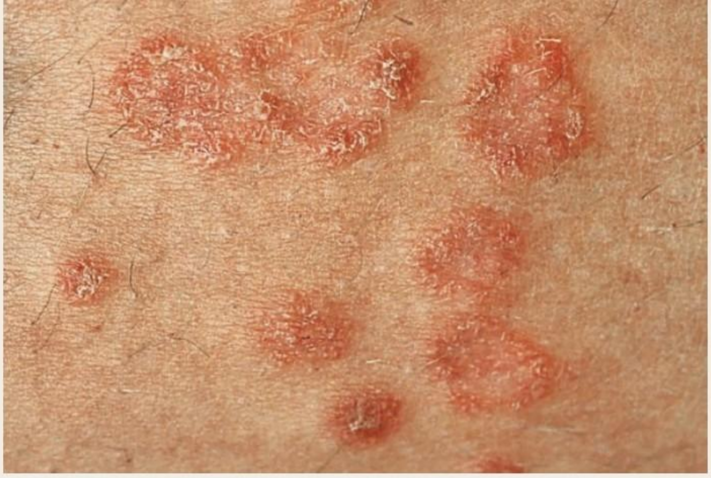


Figure 3-1. Psoriasis vulgaris Primary lesions are well-defined, reddish or salmon-pink papules, droplike, with a loosely adherent silvery-white lamellar scale.

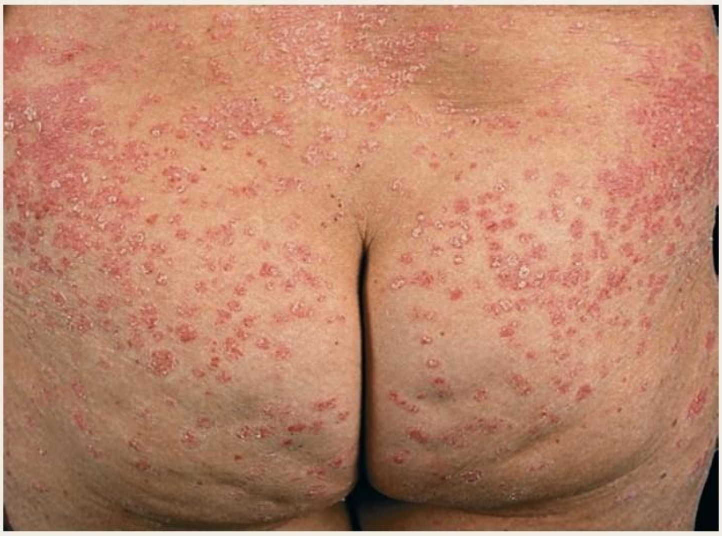


Figure 3-2. Psoriasis vulgaris: buttocks (guttate type) Small, discrete, erythematous, scaling, papules that tend to coalesce, appearing after a group A streptococcal pharyngitis. There was a family history of psoriasis.

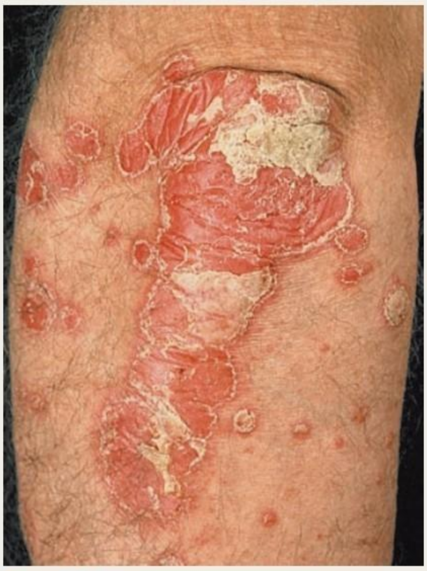


Figure 3-3. Psoriasis vulgaris: elbow Chronic stable plaque psoriasis on the elbow. In this location, scales can either accumulate to oyster shell-like hyperkeratosis, or are shed in large sheets revealing a beefy-red base. This plaque has arisen from the coalescence of smaller, papular lesions that can still be seen on lower arm.

lesion is extremely chronic, it adheres tightly resembling an oyster shell (Fig. 3-3).

Distribution and Predilection Sites

Acute Guttate. Disseminated, generalized, mainly trunk.

Chronic Stable. Single lesion or lesions localized to one or more predilection sites: elbows, knees, sacral gluteal region, scalp, and palm/soles (Fig. 3-5). Sometimes only regional involvement (scalp), often generalized.

Pattern. Bilateral, often symmetric (predilection sites, Fig. 3-5); often spares exposed areas.

Psoriasis in Skin of Color. In dark brown or black people psoriasis lacks the bright red color. Lesions are brown to black but otherwise their morphology is the same as in white skin (Fig. 3-6).

Special Sites

Palms and Soles. May be the only areas involved. There is massive silvery white or yellowish hyperkeratosis, which is not easily removed (Fig. 3-7). The inflammatory plaque at the base is always sharply demarcated (Fig. 3-7A). There may be cracking, painful fissures and bleeding.

Scalp. Plaques, sharply marginated, with thick adherent scales (Fig. 3-8). Often very pruritic. *Note:* Psoriasis of the scalp does not lead to hair loss. Scalp psoriasis may be part of generalized psoriasis or the only site involved.

This plaque has arisen from the coalescence of smaller, papular lesions that can still be seen on lower arm.

loss. Scalp psoriasis may be part of generalized psoriasis or the only site involved.

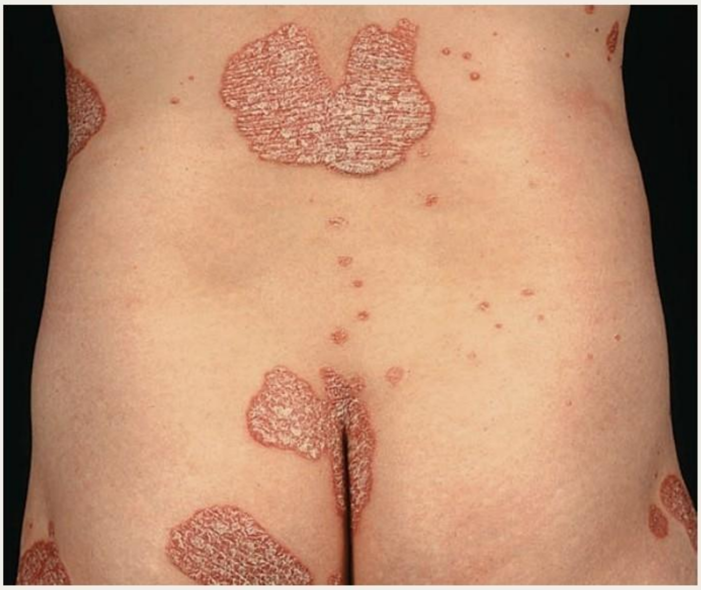


Figure 3-4. Psoriasis vulgaris: chronic stable type Multiple large scaling plaques on the trunk, buttock, and legs. Lesions are round or polycyclic and confluent forming geographic patterns. Although this is the classical manifestation of chronic stable plaque psoriasis, the eruption is still ongoing, as evidenced by the small guttate lesions in the lumbar and lower back area. This patient was cleared by acitretin/PUVA combination treatment within 4 weeks.

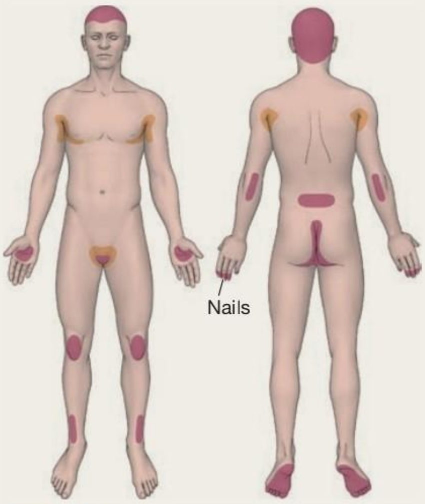


Figure 3-5. Predilection sites of psoriasis.

Face. Uncommon but when involved, usually associated with a refractory type of psoriasis (Fig. 3-9).

Chronic Psoriasis of the Perianal and Genital Regions and of the Body Folds—Inverse Psoriasis. Due to the warm and moist environment in these regions, plaques usually not scaly but macerated, often bright red and fissured (Fig. 3-10). Sharp demarcation permits distinction from intertrigo, candidiasis, and contact dermatitis.

Nails. Fingernails and toenails frequently (25%) involved, especially with concomitant arthritis (Fig. 3-11). Nail changes include pitting, subungual hyperkeratosis, onycholysis, and yellowish-brown spots under the nail plate—the *oil spot* (pathognomonic).

Laboratory Examinations

Dermatopathology

Marked overall thickening of the epidermis (acanthosis) and thinning of epidermis over

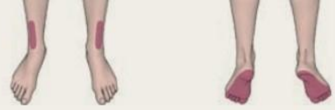


Figure 3-5. Predilection sites of psoriasis.

Laboratory Examinations

Dermatopathology

Marked overall thickening of the epidermis (acanthosis) and thinning of epidermis over

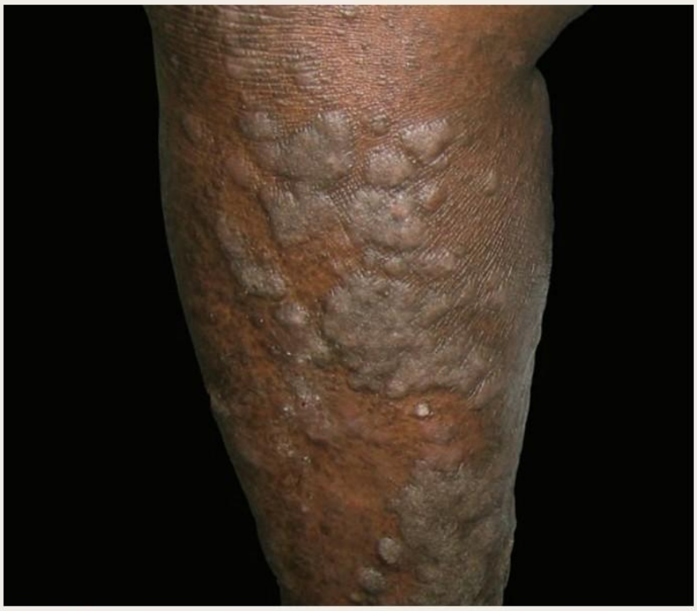


Figure 3-6. Confluent small psoriatic plaques in a 52-year-old female with HIV disease. She also had psoriatic arthritis. The lesions show less erythema than in Caucasian skin. Because the patient had been using emollients, no scale is noted.

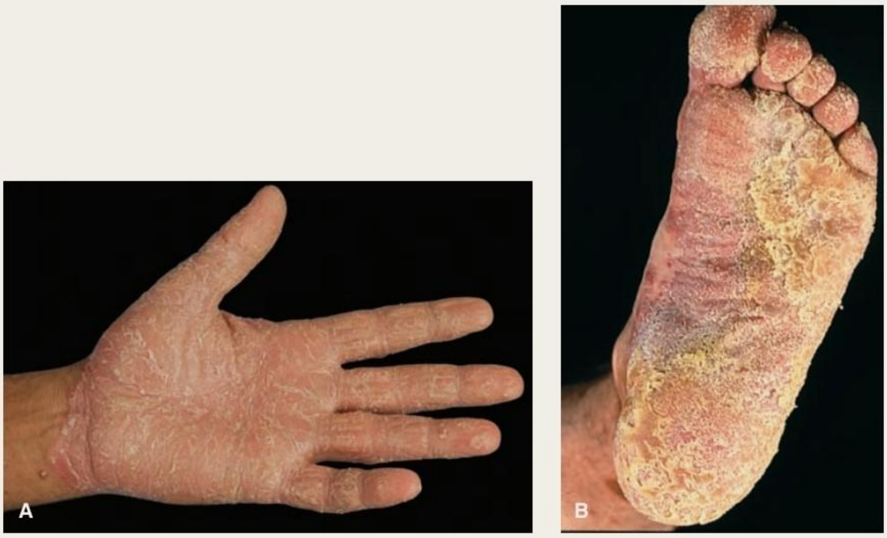


Figure 3-7. **(A). Psoriasis, palmar involvement** The entire palm is involved by large adherent scales with fissures. The base is erythematous and there is a sharp margin on the wrist. **(B) Psoriasis vulgaris: soles** Erythematous plaques with thick, yellowish, lamellar scale and desquamation on sites of pressure arising on the plantar feet. Note sharp demarcation of the inflammatory lesion on the arch of the foot. Similar lesions were present on the palms.

Figure 3-8. Psoriasis of the scalp There is massive compaction of horny material on the entire scalp. In some areas, the thick asbestos-like scales have been shed revealing a red infiltrated base. Alopecia is not due to psoriasis but is androgenetic alopecia.

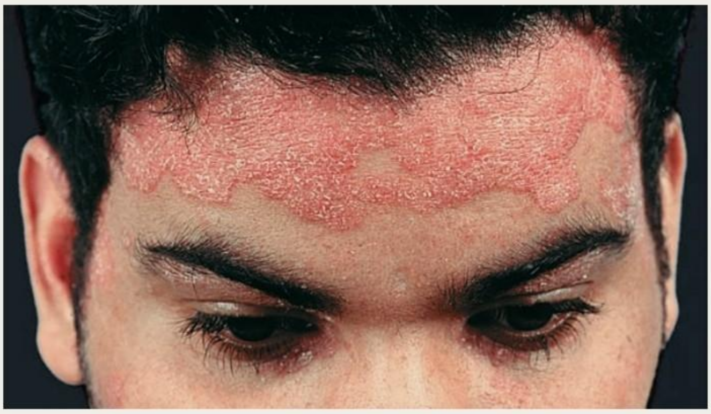


Figure 3-9. Psoriasis, facial involvement Classic psoriatic plaque on the forehead of a 21-year-old male who also had massive scalp involvement.

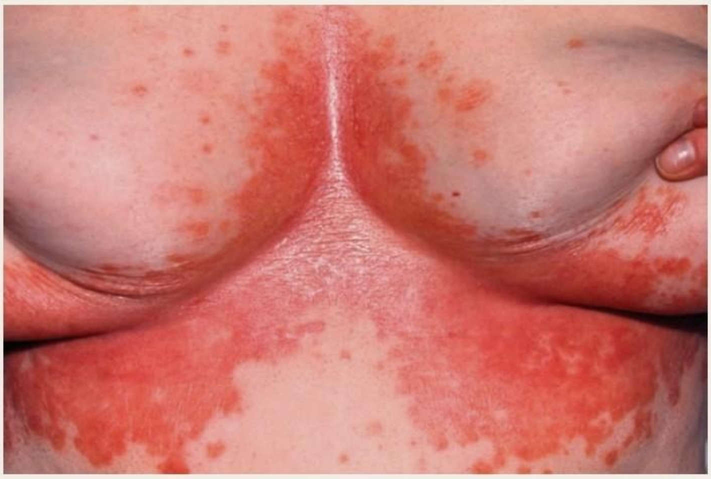


Figure 3-10. Psoriasis vulgaris: inverse pattern Because of the moist and warm environment in the submammary region, scales have been macerated and shed revealing a brightly erythematous and glistening base.

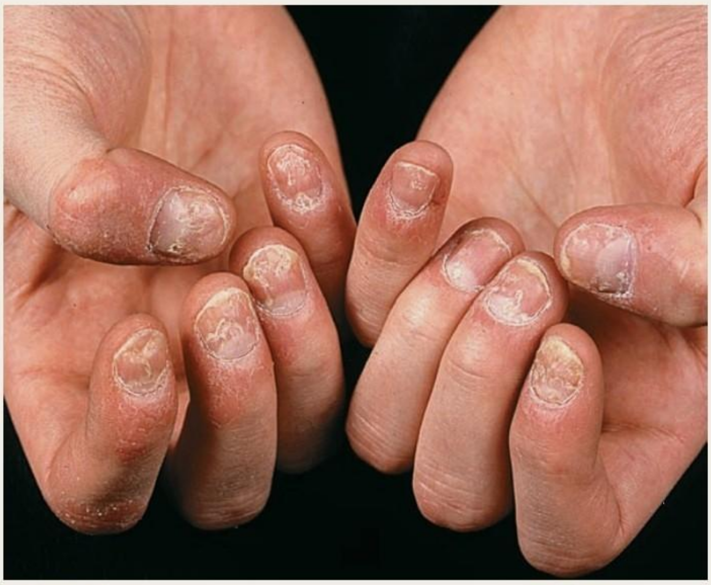


Figure 3-11. Psoriasis of the fingernails Pits have progressed to elkonyxis (holes in the nail plates), and there is transverse and longitudinal ridging. This patient also has paronychial psoriasis and psoriatic arthritis (for further images of nail involvement, see Section 34).

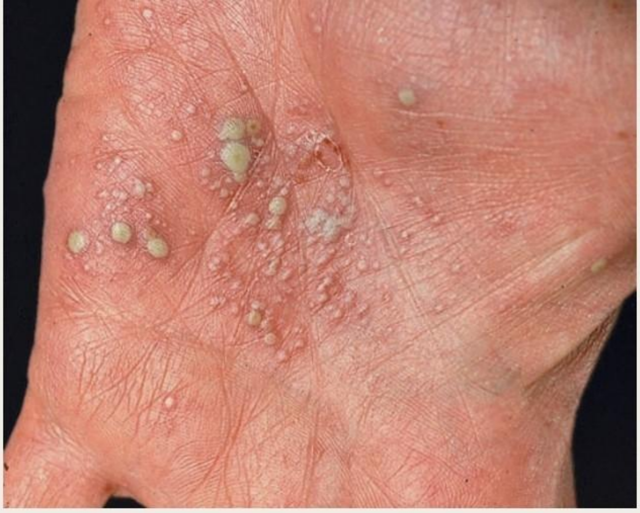
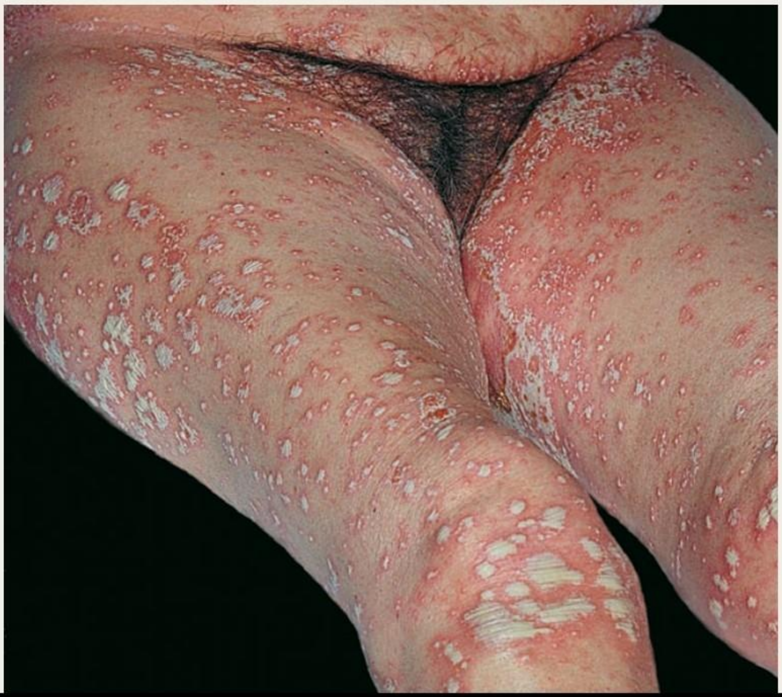


Figure 3-12. Palmar pustulosis Creamy-yellow pustules that are partially confluent on the palm of a 28-year-old female. Pustules are sterile and pruritic, and when they get larger, become painful. At the time of this eruption, there was no other evidence of psoriasis anywhere else on the body, but 2 years later the patient developed chronic stable plaque psoriasis on the trunk.

Generalized Acute Pustular Psoriasis (Von Zumbusch) ■ ○

ICD-9: 696.1 ∘ ICD-10: L40.1

- A rare life-threatening medical problem with abrupt onset.
- Burning, fiery-red erythema topped by pinpoint sterile yellow pustules in clusters spreading within hours over entire body. Coalescing lesions form “lakes” of pus (Fig. 3-13). Easily wiped off.
- Waves of pustules follow each other.
- Fever, malaise, and leukocytosis.
- Symptoms: burning, painful; patient appears frightened.
- Onycholysis and shedding of nails; hair loss of the telogen defluvium type (see Section 33), 2–3 months later; circinate desquamation of tongue.
- Pathogenesis unknown. Fever and leukocytosis result from release of cytokines and chemokines into circulation.
- Differential diagnosis: pustular drug eruption (see Section 23); generalized HSV infection.
- May follow, evolve, or be followed by psoriasis vulgaris.
- Special types: *Impetigo herpetiformis*: generalized pustular psoriasis in pregnant woman with hypocalcemia. *Annular type*: in children with less constitutional symptoms (Fig. 3-14A). *Psoriasis cum pustulatione* (psoriasis vulgaris with pustulation: In maltreated psoriasis vulgaris. No constitutional symptoms. *Acrodermatitis continua of Hallopeau*: Chronic recurrent pustulation of nail folds, nail beds, and distal fingers leading to nail loss (Fig. 3-14B). Occurs alone or with generalized pustular psoriasis.



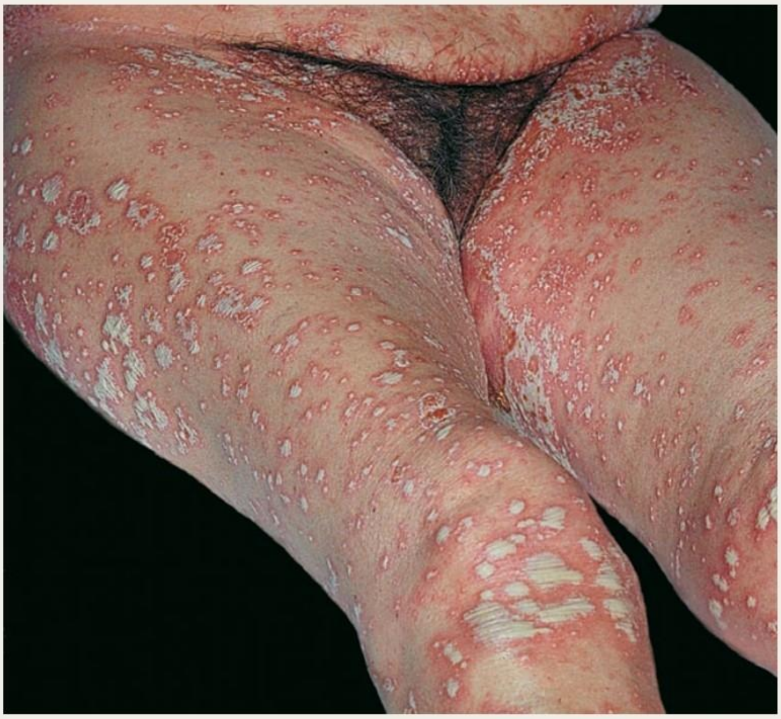


Figure 3-13. Generalized acute pustular psoriasis (von Zumbusch) This female patient was toxic and had fever and peripheral leukocytosis. The entire body was covered with showers of creamy-white coalescing pustules on a fiery-red base. Since these pustules are very superficial, they can be literally wiped off, which results in red oozing erosions.

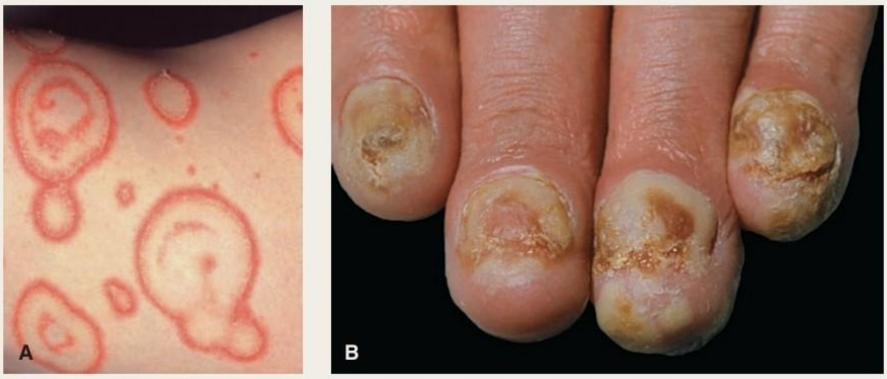


Figure 3-14. (A). Anular pustular psoriasis This occurs mainly in children and consists of expanding ring-like micropustular eruptions on a highly inflammatory base that is clear in the center and results in a collarette-like scaling at the margin. There is hardly any systemic toxicity. **(B) Acrodermatitis continua** of Hallopeau with acral pustule formation, subungual lakes of pus, and destruction of nail plates. This may lead to permanent loss of nails and scarring.

Psoriatic Erythroderma ICD-9: 696.1 ◦ ICD-10: L40 ◧ ○

In this condition psoriasis involves the entire skin. See Section 8.

Psoriatic Arthritis ICD-9: 696.0 ◦ ICD-10: L40.5 ◧ ◐

- Seronegative. Psoriatic arthritis is included among the seronegative spondyloarthropathies, which include ankylosing spondylitis, enteropathic arthritis, and reactive arthritis.
- Asymmetric peripheral joint involvement of upper extremities and especially distal
- Mutilating with bone erosions, osteolysis, or ankylosis. Telescoping fingers. Functional impairment.
- Often associated with psoriasis of nails (Figs. 3-11 and 3-15).
- Associated with MHC class I antigens, while

hood, middle age, >60 years.

2. Type of psoriasis: guttate, plaque, palmar and palmopustular, generalized pustular psoriasis, erythrodermic psoriasis.
3. Site and extent of involvement: *localized* to palms and soles, scalp, anogenital area, scattered plaques but <5% involvement; *generalized* and >30% involvement.
4. Previous treatment: ionizing radiation, systemic glucocorticoids, photochemotherapy (PUVA), cyclosporine (CS), and methotrexate (MTX).
5. Associated medical disorders (e.g., HIV disease).

Management of psoriasis is discussed in the context of types of psoriasis, sites, and extent of involvement. Psoriasis has to be managed by a dermatologist.

Localized Psoriasis (see Fig. 3-3)

- *Topical fluorinated glucocorticoid* covered with plastic wrap. Glucocorticoid-impregnated tape also useful. Beware of glucocosteroid side effects.

tonide aqueous suspension 3 mg/mL diluted with normal saline injected *intradermally* into lesions. Beware of hypopigmentation in skin of color.

- Topical *anthralin* also effective but can be irritant.
- *Vitamin D analogues* (calcipotriene, ointment and cream) are good nonsteroidal antipsoriatic topical agents but less effective than corticosteroids; they are not associated with cutaneous atrophy; can be combined with corticosteroids. Topical tacrolimus, 0.1%, similarly effective.
- Topical pimecrolimus, 1%, is effective in inverse psoriasis and seborrheic dermatitis-like psoriasis of the face and ear canals.
- *Tazarotene* (a topical retinoid, 0.05 and 0.1% gel) has similar efficacy, best combined with class II topical glucocorticoids.
- All these topical treatments can be combined with 311-nm UVB phototherapy or PUVA.

Scalp. Superficial scaling and lacking thick plaques: Tar or ketoconazole shampoos *followed*

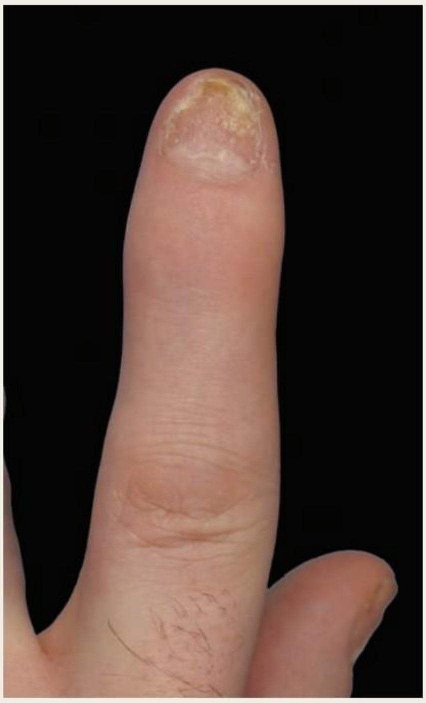


Figure 3-15. Psoriatic arthritis Dactylitis of index finger. Note sausage-like thickening over interphalangeal joints. There is psoriasis of the nail.

by betamethasone valerate, 1% lotion; if refractory, clobetasol propionate, 0.05% scalp application. In thick, adherent plaques (Fig. 3-8): scales have to be removed by 10% salicylic acid in mineral oil, covered with a plastic cap and left on overnight before embarking on topical therapy. If this is unsuccessful, consider systemic treatment (see below).

Palms and Soles (Fig. 3-7). Occlusive dressings with class I topical *glucocorticoids*. If ineffective, *PUVA* either systemically or as *PUVA* soaks (immersion in 8-methoxypsoralen solution and subsequent UVA exposure). Retinoids (acitretin > isotretinoin), orally, removes the thick hyperkeratosis of the palms and soles; however, combination with topical glucocorticoids or PUVA (re-PUVA) is much more efficacious. Systemic treatments should be considered.

Palmoplantar Pustulosis (Fig. 3-12). PUVA *soaks* and glucocorticosteroids are effective. Systemic

these are atrophy-prone regions; steroids should be applied for only limited periods of time); switch to topical vitamin D derivatives or tazarotene or topical tacrolimus or pimecrolimus. If resistant or recurrent, consider systemic therapy.

Nails (Fig. 3-11). Topical treatments of the fingernails are unsatisfactory. Systemic MTX and CS therapy effective but takes time and thus prone to side effects.

Generalized Psoriasis

Acute, Guttate Psoriasis (Fig. 3-2). Treat streptococcal infection with antibiotics. Narrowband UVB irradiation most effective.

Generalized Plaque-Type Psoriasis (Fig. 3-4). PUVA or systemic treatments that are given as either mono—or combined—or rotational therapy. Combination therapy denotes the combination of two or more modalities, while rotational therapy denotes switching the patient after clearing and a subsequent relapse to another different treatment.

Narrowband UVB Phototherapy (311 nm). Effective only in thin plaques; effectiveness is increased by combination with topical glucocorticoids, vitamin D analogues, tazarotene, or topical tacrolimus/pimecrolimus.

Oral PUVA. Treatment consists of oral ingestion of 8-methoxypsoralen (8-MOP) (0.6 mg 8-MOP per kilogram body weight) or, in some European countries, 5-MOP (1.2 mg/kg body weight) and exposure to doses of UVA that are adjusted to the sensitivity of the patient. Most patients clear after 19–25 treatments, and the amount of UVA needed ranges from 100 to 245 J/cm^2.

Long-Term Side Effects. PUVA keratoses and squamous cell carcinomas in some patients who receive an excessive number of treatments.

Oral Retinoids. Acitretin and isotretinoin are effective in inducing desquamation but only moderately effective in clearing psoriatic plaques. Highly effective when combined with 311-nm UVB or PUVA (called re-PUVA). *The latter is in fact the most effective therapy to date for generalized plaque psoriasis.*

Methotrexate Therapy. Oral MTX is one of the most effective treatments but response is slow and long-term treatment is required. Hepatic toxicity may occur after cumulative doses in normal persons (≥1.5 g).

The Triple-Dose (Weinstein) Regimen. Preferred

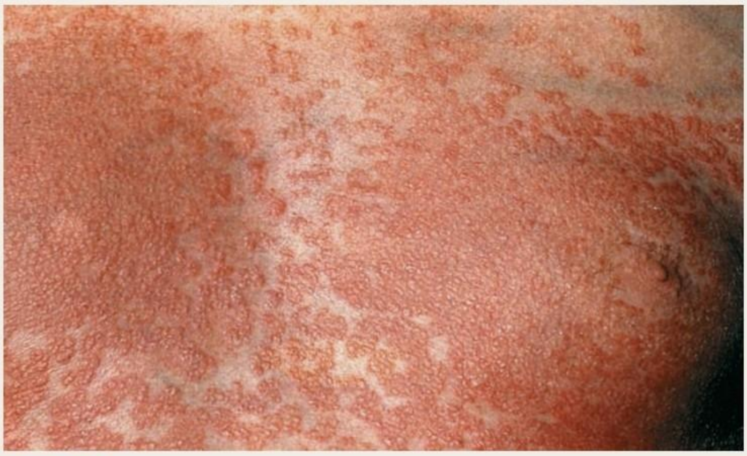


Figure 3-16. Pityriasis rubra pilaris (type 1, classic adult) Orange-red follicular papules beginning on the head and neck have coalesced on the chest of a 57-year-old male. There are sharply demarcated islands of unaffected normal skin.

Management

Topical therapies consist of emollients, keratolytic agents, vitamin D_3 (calcipotriol), glucocorticoids, and vitamin A analogues (tazarotene). All are not very effective. Phototherapy (ultraviolet A phototherapy, narrowband ultraviolet B phototherapy, and photochemotherapy) is effective in some cases. Most effective treatment consists of systemic administration of MTX or retinoids (both as in psoriasis). In type 6: HAART. The anti-TNF agents, e.g., infliximab and etanercept are effective.

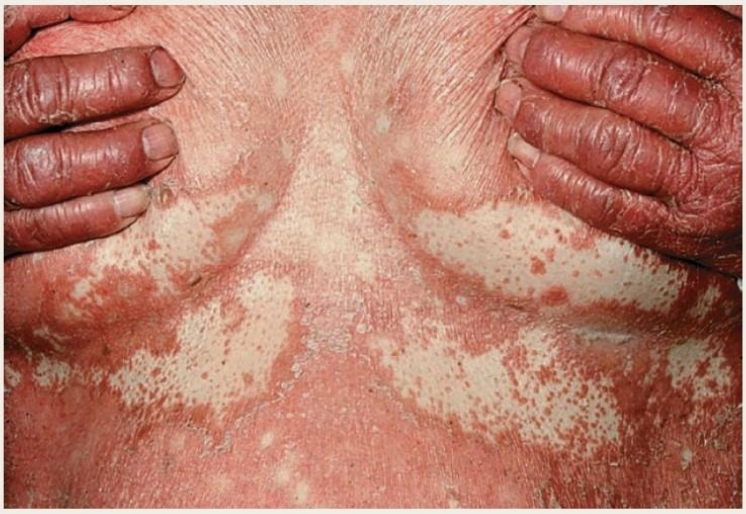
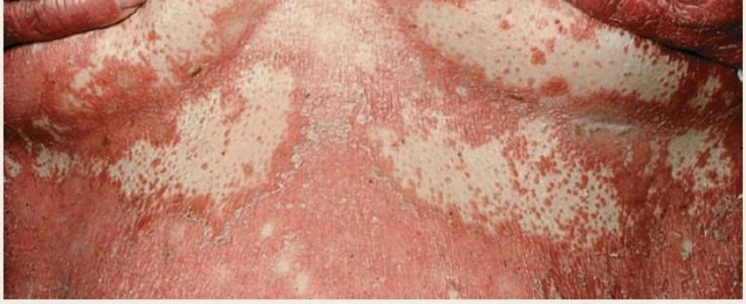


Figure 3-17. Pityriasis rubra pilaris (type 1, classic adult) Orange-reddish papules have coalesced to near erythroderma, sparing isolated islands of normal skin. Also note involvement of the hands in this 55-year-old woman.



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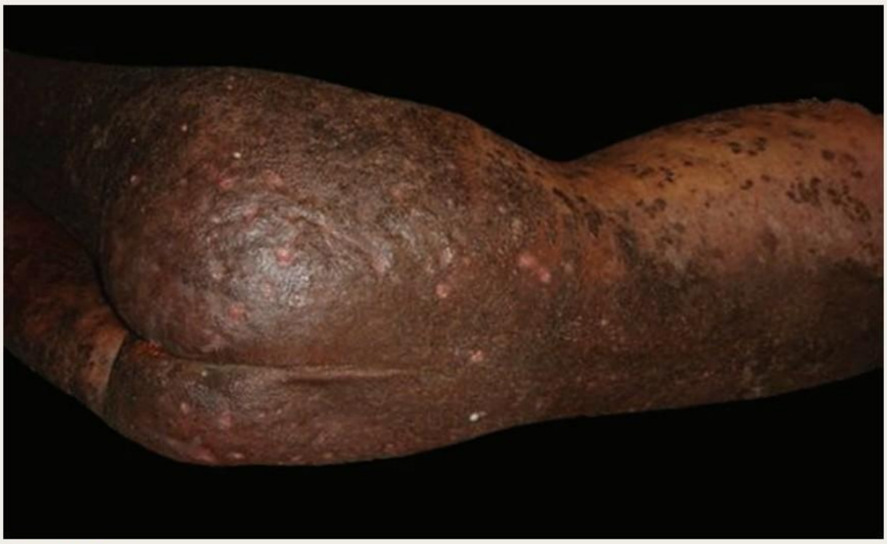


Figure 3-18. Pityriasis rubra pilaris in black skin Here papules do not have the classical orange color seen in Caucasians but are brown and therefore pose a diagnostic problem. Their shape and distribution and the areas of spared normal skin are diagnostic clues.

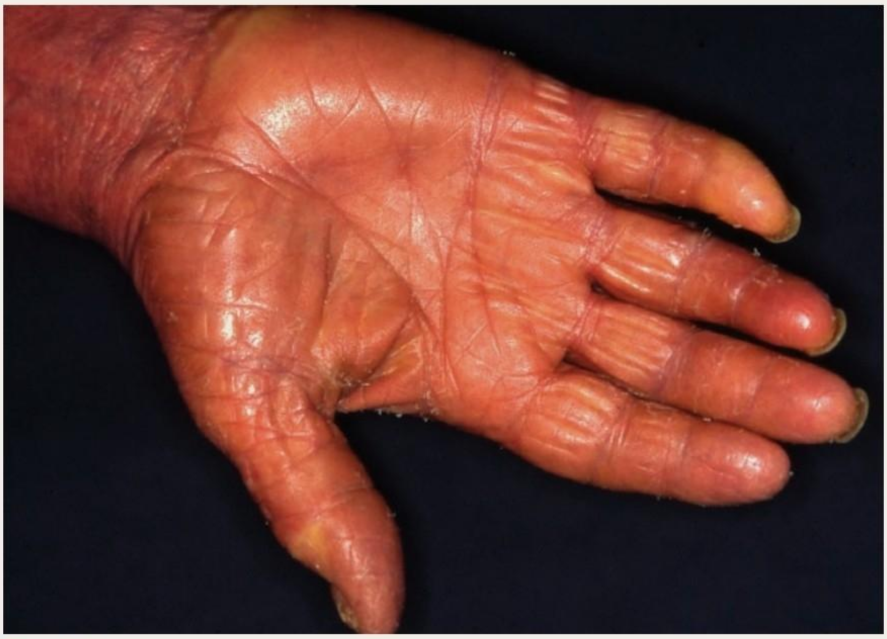


Figure 3-19. Pityriasis rubra pilaris on palms There is diffuse, waxy hyperkeratosis with an orange hue.

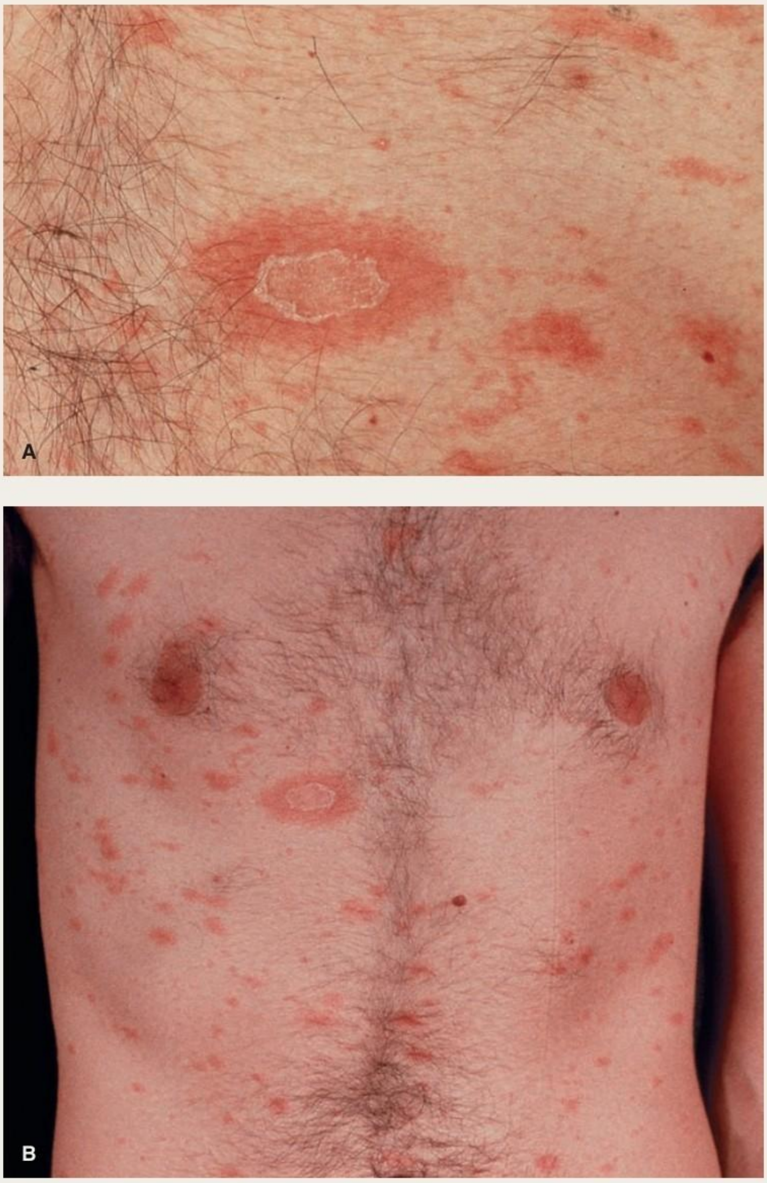
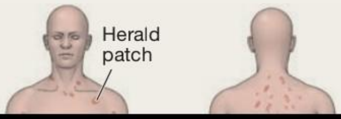


Figure 3-20. **Pityriasis rosea** **(A).** Herald patch. An erythematous (salmon-red) plaque with a collarette scale on the trailing edge of the advancing border. Collarette means that scale is attached at periphery and loose toward the center of the lesion. **(B)** Overview of exanthem of pityriasis rosea with the herald patch shown in part **(A).** There are papules and small plaques with oval configurations that follow the lines of cleavage. The fine scaling of the salmon-red papules cannot be seen at this magnification, while the collarette of the herald patch is obvious.

Figure 3-21. **Pityriasis rosea** Distribution "Christmas tree" pattern on the back.



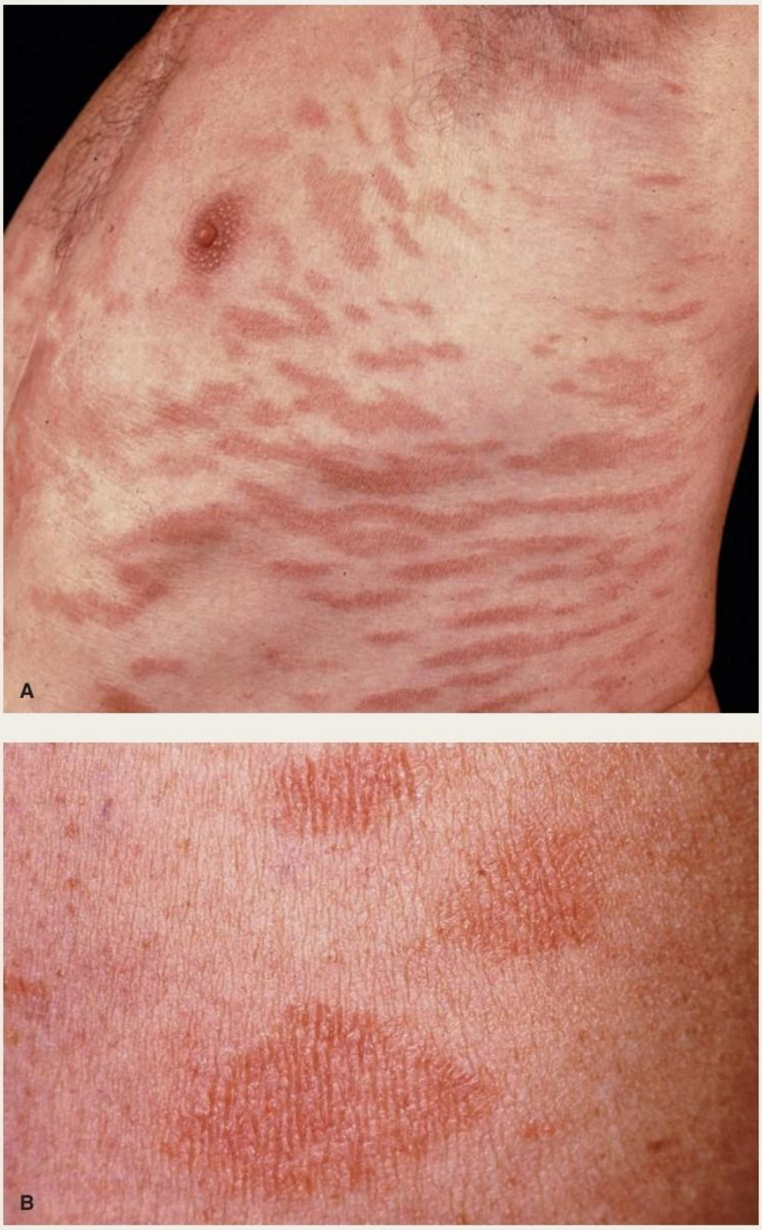
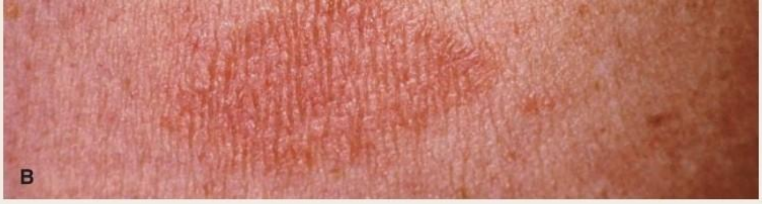


Figure 3-22. **Digitate dermatosis (small-plaque parapsoriasis) (A).** The lesions are asymptomatic, yellowish or fawn-colored, very thin, well defined, slightly scaly and superficially wrinkled patches. They are oval and follow the lines of cleavage of the skin, giving the appearance of a "hug" that left fingerprints on the trunk. The long axis of these lesions often reaches more than 5 cm. **(B)** Close up of smaller lesions showing wrinkling of surface.



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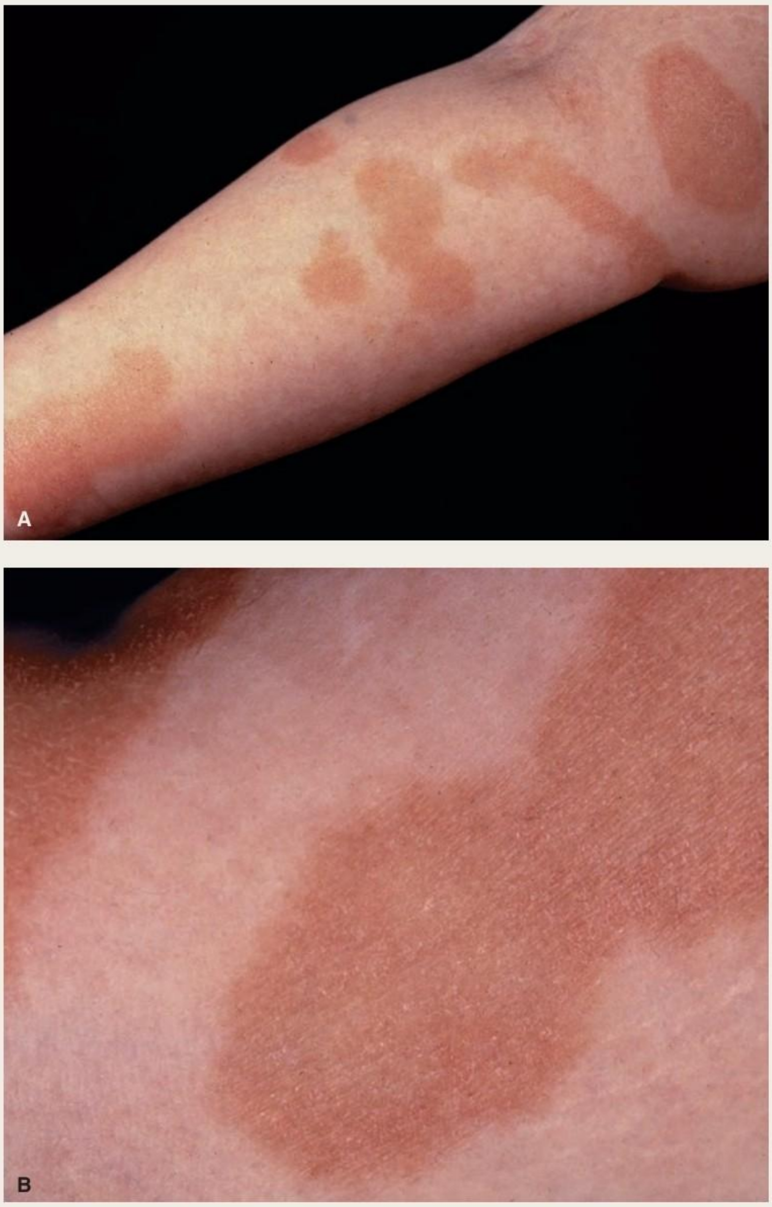


Figure 3-23. Large-plaque parapsoriasis (parapsoriasis en plaques) (A) The lesions are asymptomatic, well-defined, rounded, slightly scaly, thin plaques, or patches. The lesions can be larger than 10 cm and are light red-brown or salmon-pink. There may be atrophy in some areas. The lesions here are located on the extremities, but they are more commonly noted on the trunk. These lesions must be carefully followed, and repeated biopsies are necessary to detect mycosis fungoides. This entity may be considered as a prestage of mycosis fungoides. **(B)** Close up of lesions showing minimal scaling and wrinkled surface.

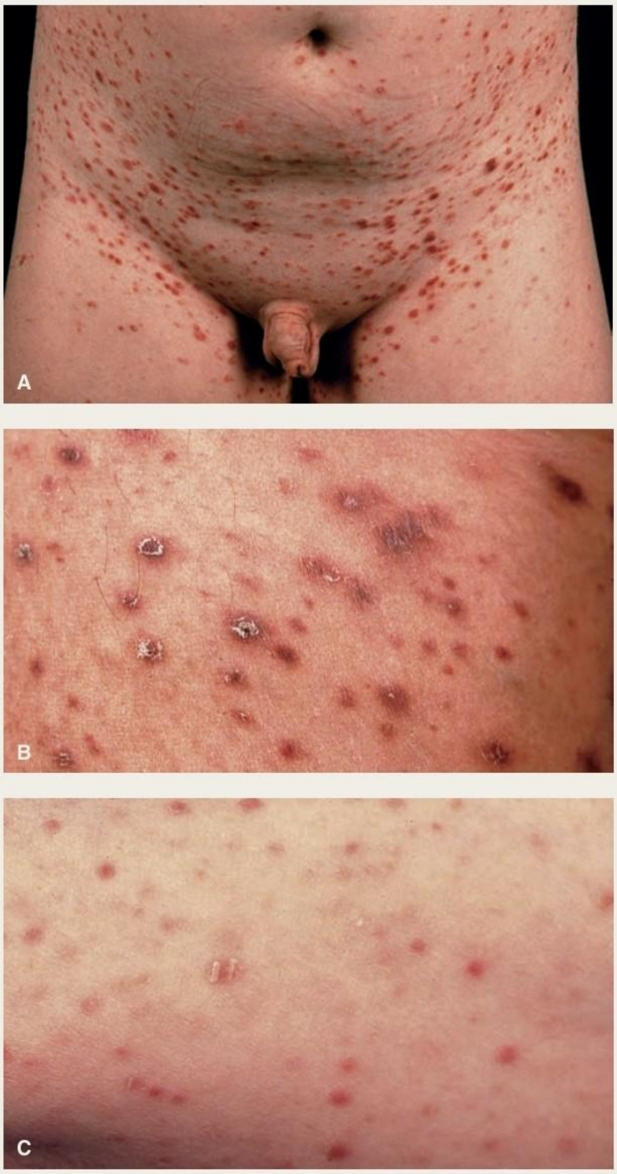


Figure 3-24. **Pityriasis lichenoides et varioliformis acuta (PLEVA) (A)** Randomly distributed red papules of different size, some of which show hemorrhagic crusting. In this 5-year-old child, the eruption appeared in crops over a period of 10 days. **(B) PLEVA** lesions in a 38-year-old Indonesian man. Lesions are more hyperpigmented and there is considerable scaling and crusting. **(C) Pityriasis lichenoides chronica (PLC)** Discrete papules with fine mica-like scales that become more visible after slight scraping. In contrast to PLEVA, there is no hemorrhagic crusting.

SECTION 4

Sites of Predilection. Typical and almost diagnostic: extensor areas—elbows (Fig. 6-18), knees. Strikingly symmetrical. Buttocks, scapular, and

allergic contact dermatitis, atopic dermatitis, scabies, neurotic excoriations, papular urticaria, and bullous autoimmune disease (see Table 6-3).

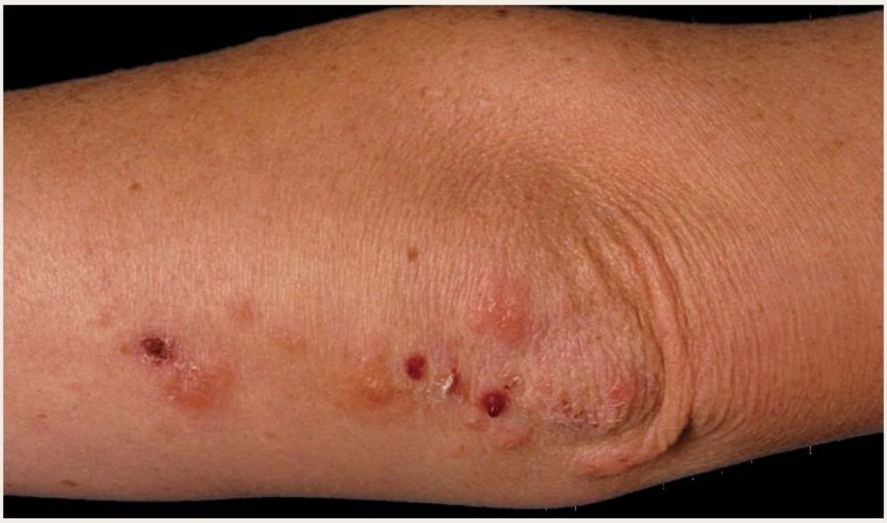


Figure 6-18. Dermatitis herpetiformis These are the classic early lesions. Papules, urticarial plaques, small grouped vesicles, and crusts on the elbow of a 23-year-old male.

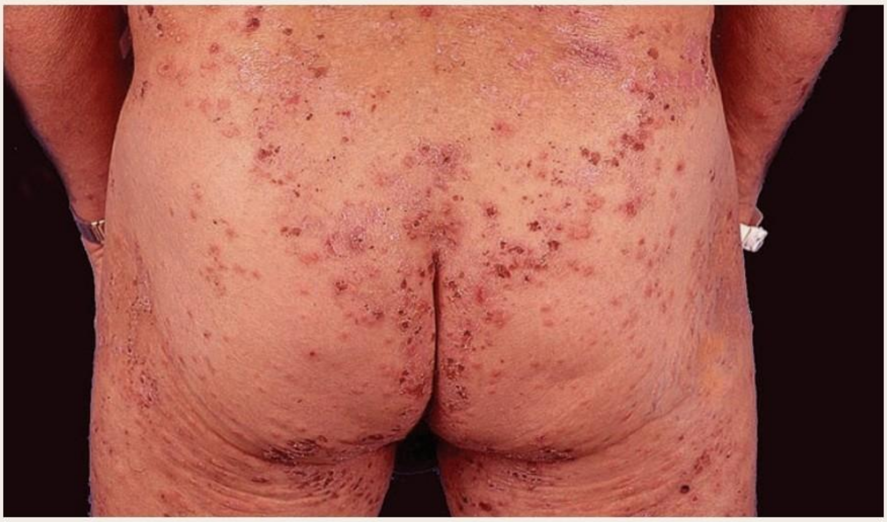
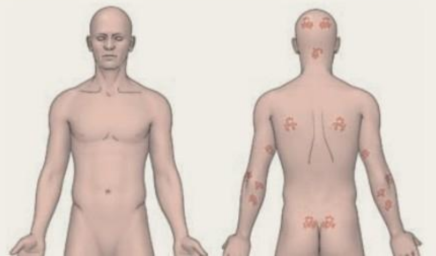


Figure 6-19. Dermatitis herpetiformis A 56-year-old male patient with a generalized highly pruritic eruption. The diagnosis can be made upon first sight by the distribution of the lesions. Most heavily involved are the sacral and gluteal areas (note butterfly-like distribution) and (not seen in this picture) the knees, elbows; the scapular areas. Upon close inspection, there are grouped papules, small vesicles, crusts, and erosions on an erythematous base and there is postinflammatory hypo- and hyperpigmentation.



Course

Prolonged, for many years, with a third of the patients eventually having a spontaneous remission.

Management

Systemic Therapy. Dapsone. 100–150 mg daily, with gradual reduction to as low as 50 mg twice a week. Dramatic response, often within only rarely on the face and on the neck.

Laboratory Examinations

Hematology. Elevated ESR and C-reactive protein; leukocytosis.

Bacterial Culture. Culture throat for group A β-hemolytic streptococcus, stool for *Yersinia*.

Imaging. Radiologic examination of the chest and gallium scan are important to rule out or prove sarcoidosis.

Dermatopathology. Acute (polymorphonuclear) and chronic (granulomatous) inflammation

arteritis nodosa, nodular vasculitis, pretibial myxedema, nonulcerated gumma, and lymphoma.

Management

Symptomatic. Bed rest or compressive bandages (lower legs), wet dressings.

Anti-inflammatory Treatment. Salicylates, nonsteroidal anti-inflammatory drugs. Systemic glucocorticoids—response is rapid, but their use is indicated only when the etiology is known and infectious agents are excluded.

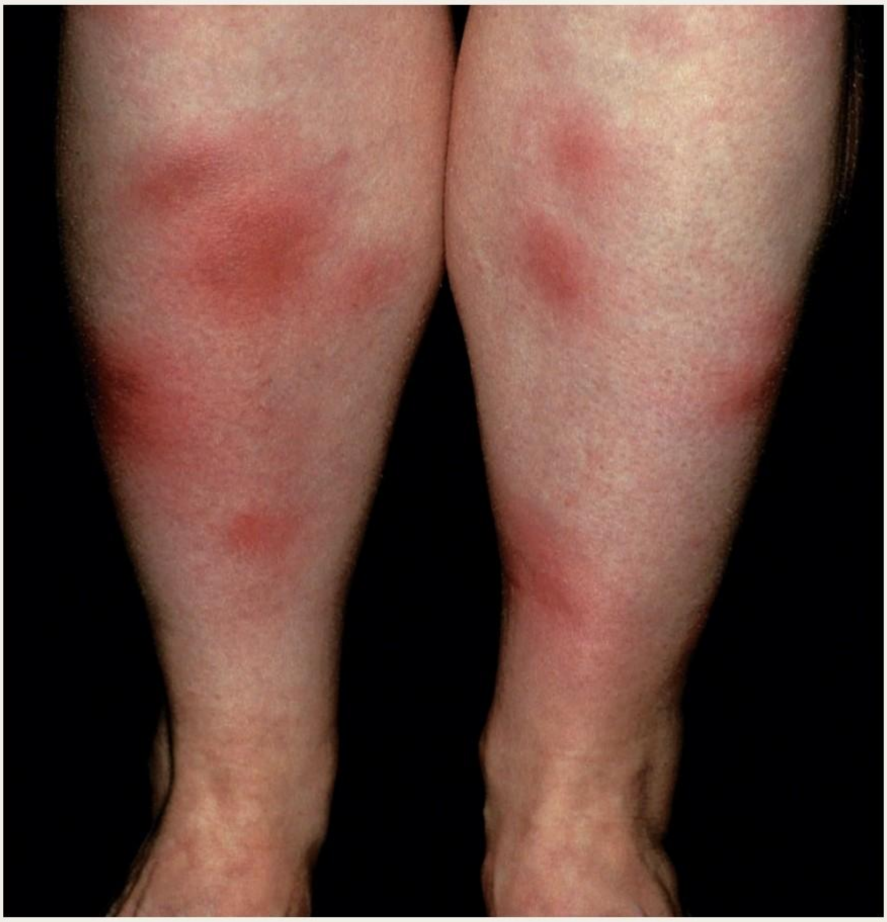


Figure 7-9. Erythema nodosum Indurated, very tender, inflammatory nodules mostly in the pretibial region. Lesions are seen as red, ill-defined erythemas but palpated as deep-seated nodules, hence the designation. In this 49-year-old female, there was also fever and arthritis of the ankle joints following an upper respiratory tract infection. The throat cultures yielded β-hemolytic streptococci.

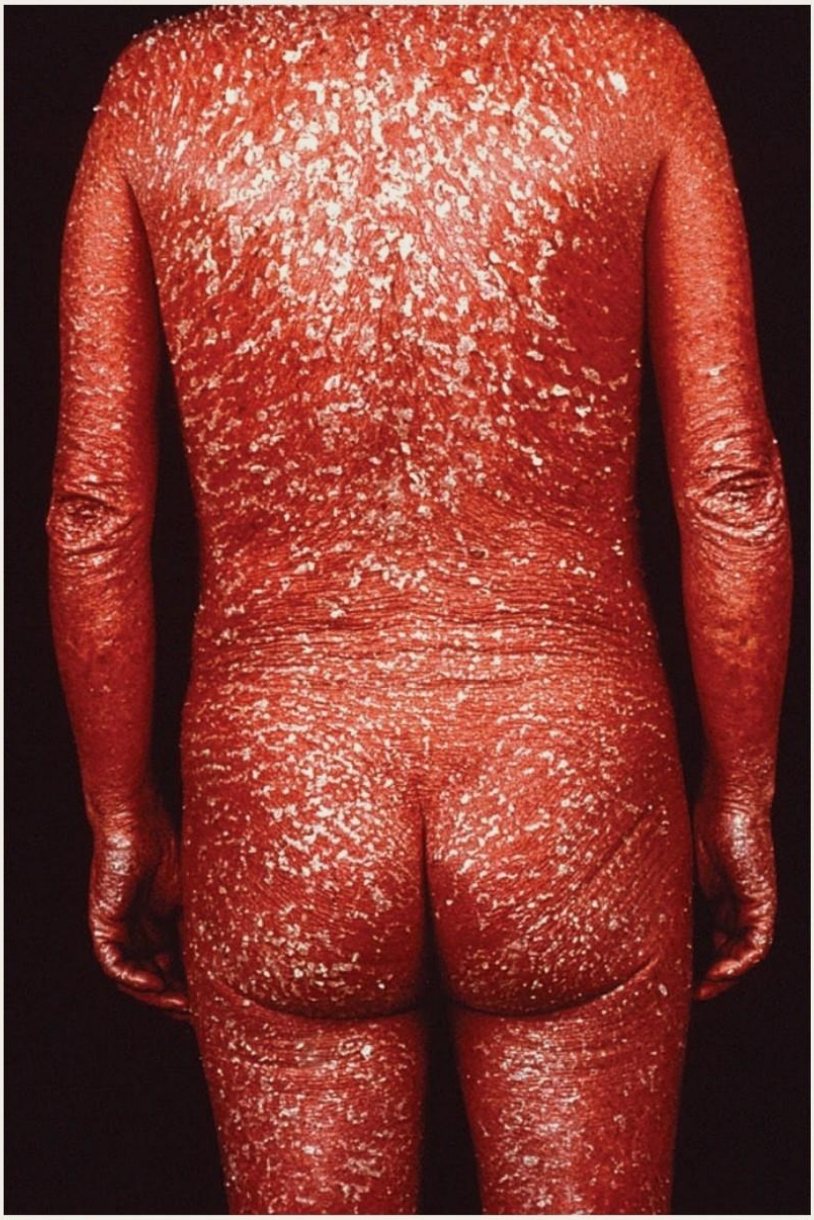


Figure 8-1. Exfoliative dermatitis: psoriasis There is universal erythema, thickening of the skin, and heavy scaling. This patient had psoriasis as suggested by the large silvery white scales and the scalp and nail involvement not seen in this illustration. The patient had fatigue, weakness, malaise, and was shivering. It is quite obvious that such massive scaling can lead to protein loss and the maximal dilatation of skin capillaries to considerable heat dissipation and high-output cardiac failure.

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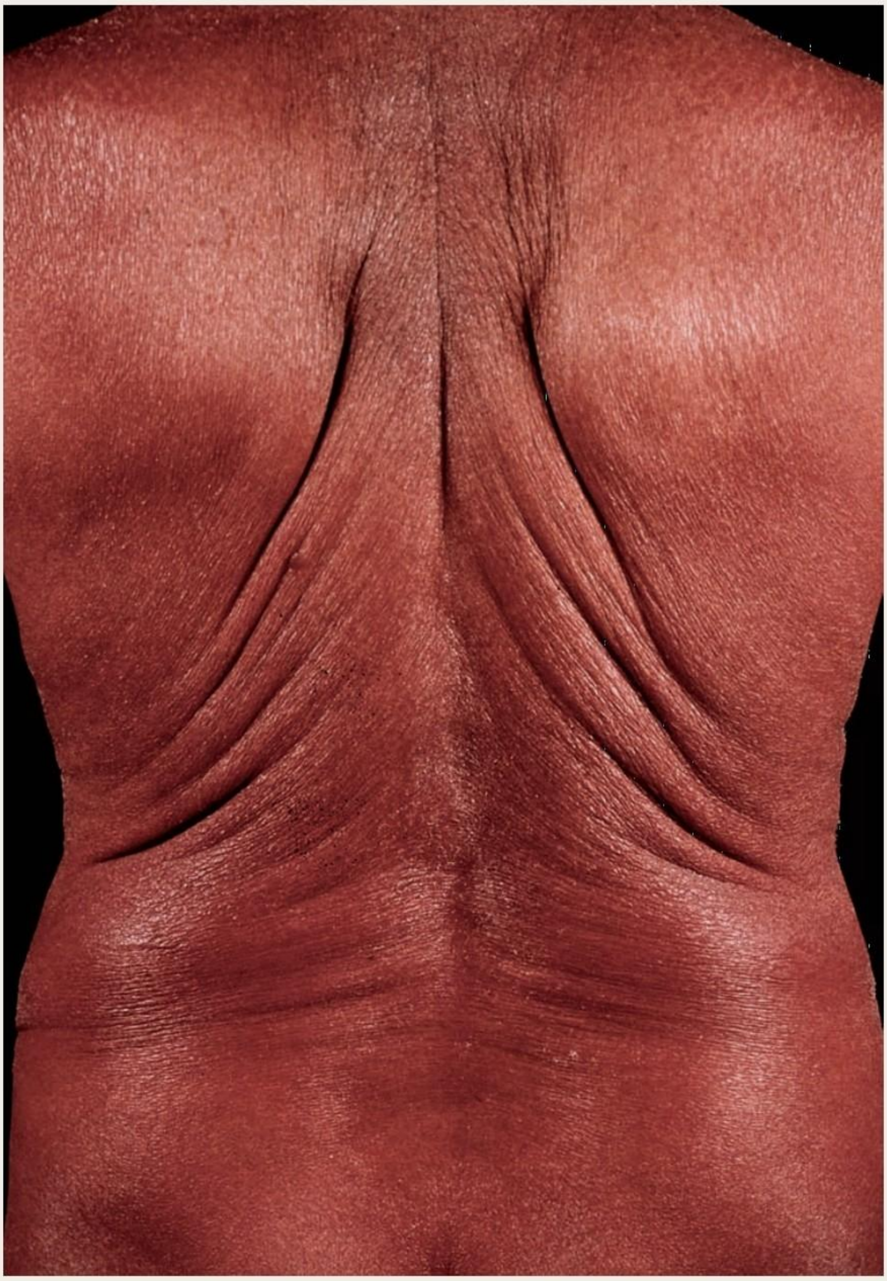

Figure 8-2. **Exfoliative dermatitis: drug induced** This is generalized erythroderma with thickening of skin resulting in increased skin folds, universal redness, a fine brawny scaling. This patient had developed erythroderma following the injection of gold salts for rheumatoid arthritis.

The diagnosis of skin eruptions is based mainly on precise identification of the type of skin lesions

into five main categories according to the type of lesion (Table 8-3).

التقويم

الحدث

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2. *Viral culture,* negative stain (electron microscopy), polymerase chain reaction for infections with herpes viruses, direct fluorescence (DIF) technique.
3. *Gram stain of aspirates or scraping of pustules.* Organisms can be seen in the lesions of acute meningococcemia, rarely in the skin lesions of gonococcemia and ecthyma gangrenosum.
4. *Touch preparation.* Helpful in deep fungal infections and leishmaniasis. The dermal

rapid serologic tests for syphilis, and serology for lupus erythematosus. Examination of urine sediment may reveal red cell casts in renal involvement in allergic vasculitis.

7. *Dark-field examination.* In the skin lesions of secondary syphilis, repeated examinations of papules show *Treponema pallidum.* Not reliable in the mouth because of resident nonpathogenic organisms but a lymph node aspirate can be subjected to dark-field examination.

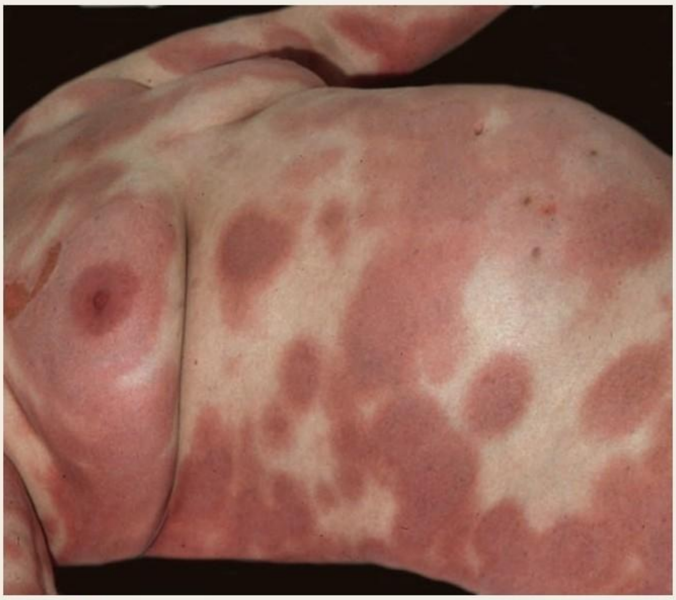
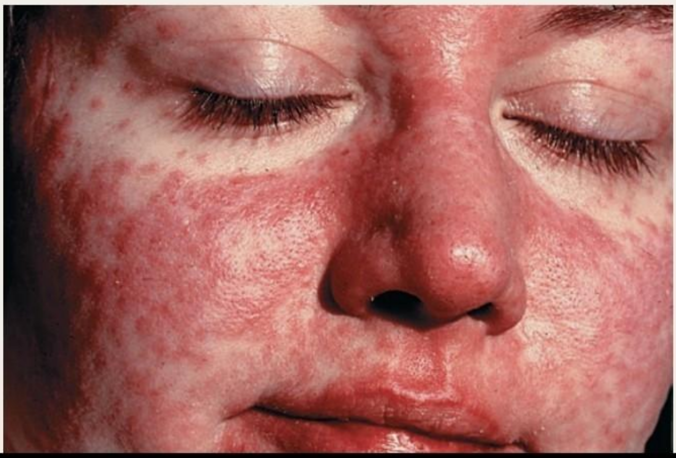


Figure 8-4. Generalized fixed drug eruption: tetracycline. Prostrated, 59-year-old woman with fever. Multiple confluent violaceous red erythematous areas, some of which later became bullous.



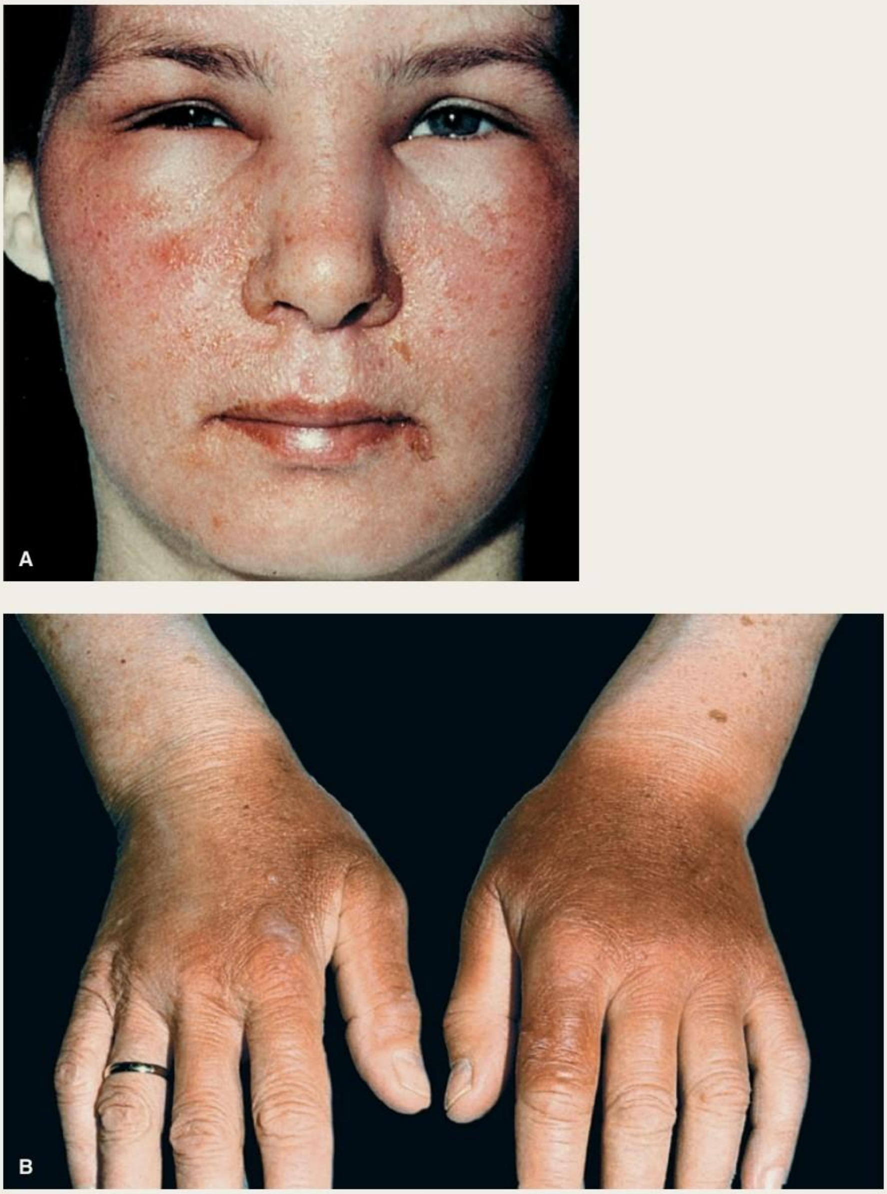


Figure 10-3. **Phototoxic drug-induced photosensitivity (A)** Massive edema and erythema in the face of a 17-year-old girl who was treated with demethylchlortetracycline for acne. **(B)** Dusky erythema with blistering on the dorsa of both hands in a patient treated with piroxicam.

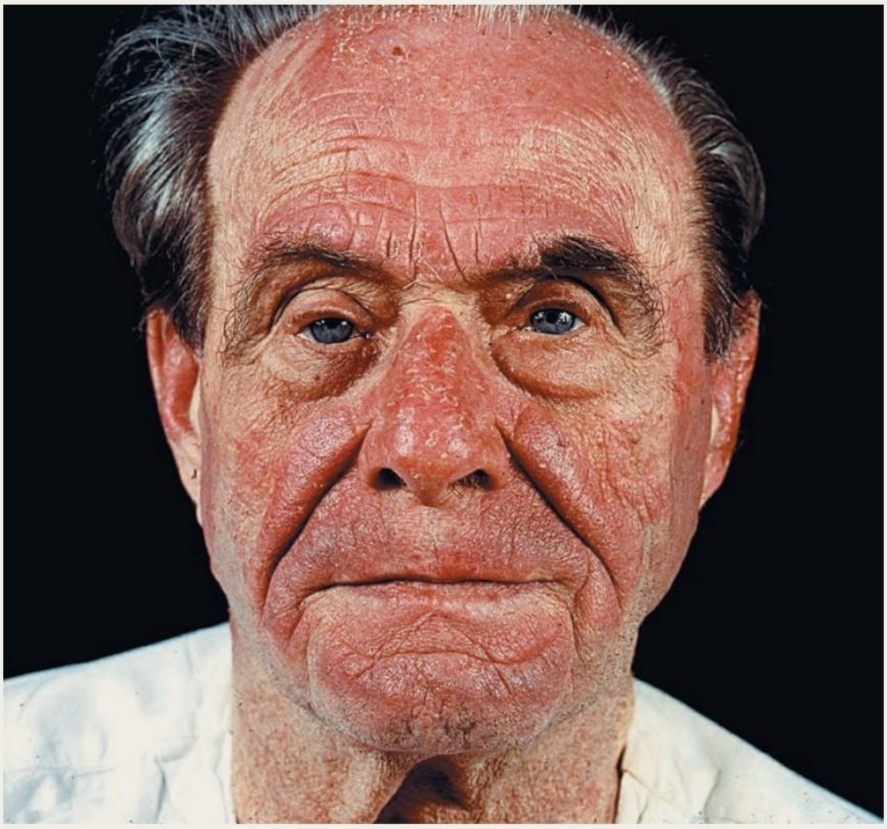
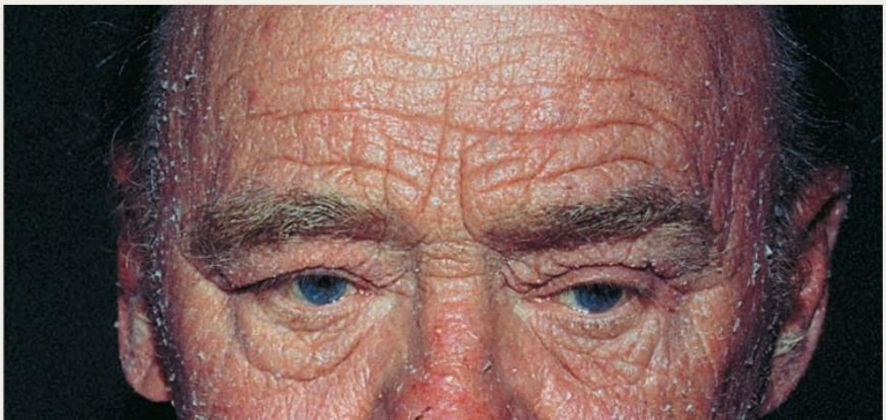


Figure 10-7. Photoallergic drug-induced photosensitivity This 60-year-old male shows an eczematous dermatitis in the face. He was taking trimethoprim–sulfamethoxazole. Note relative sparing of eyelids (protected by sunglasses), under the nose, and the area under the lower lip (shaded areas).

reaction) (Fig. 10-8). In *chronic actinic dermatitis*, the action spectrum usually broadens to involve also UVB, and the condition persists despite discontinuation of the causative photoallergen, with each new UV exposure aggravating the condition. Chronic eczema-like lichenified and extremely itchy confluent plaques result (Fig. 10-8), which lead to disfigurement and a distressing situation for the patient. As the condition is now independent of the original photoallergen and is aggravated by each new solar exposure, avoidance of photoallergen does not cure the disease.

Management

In severe cases, immunosuppression (azathioprine plus glucocorticoids or oral cyclosporine) is required.



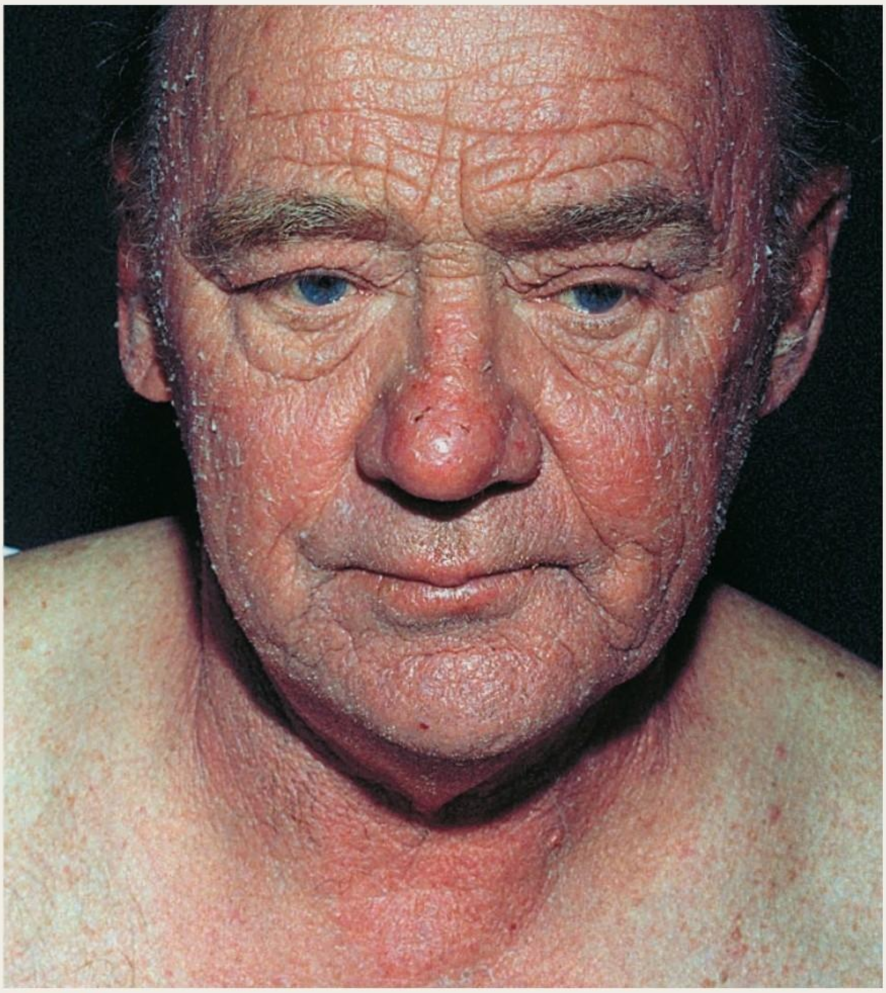


Figure 10-8. Drug-induced photosensitivity: chronic actinic dermatitis (formerly persistent light eruption) Erythematous plaques confined to the face and neck, sparing the shoulders. This male has excruciating pruritus.

ICD-9: 692.72 ◦ ICD-10: L56.4

- PMLE is a term that describes a group of

vesicles. However, in each patient, the eruption is cells.

3. The *self-destruct hypothesis* suggests that melanocytes are destroyed by toxic substances formed as part of normal melanin biosynthesis.

Clinical Manifestation

Many patients attribute the onset of their vitiligo to physical trauma (where vitiligo appears

in diameter (Figs. 13-2 and 13-3). "Chalk" or pale white, sharply marginated. The disease progresses by gradual enlargement of the old macules or by development of new ones. Margins are *convex*. Trichrome vitiligo (three colors: white, light brown, dark brown) represents different stages in the evolution of vitiligo. Pigmentation around a hair follicle in a white macule represents residual pigmentation or return of pigmentation (Fig. 13-3).

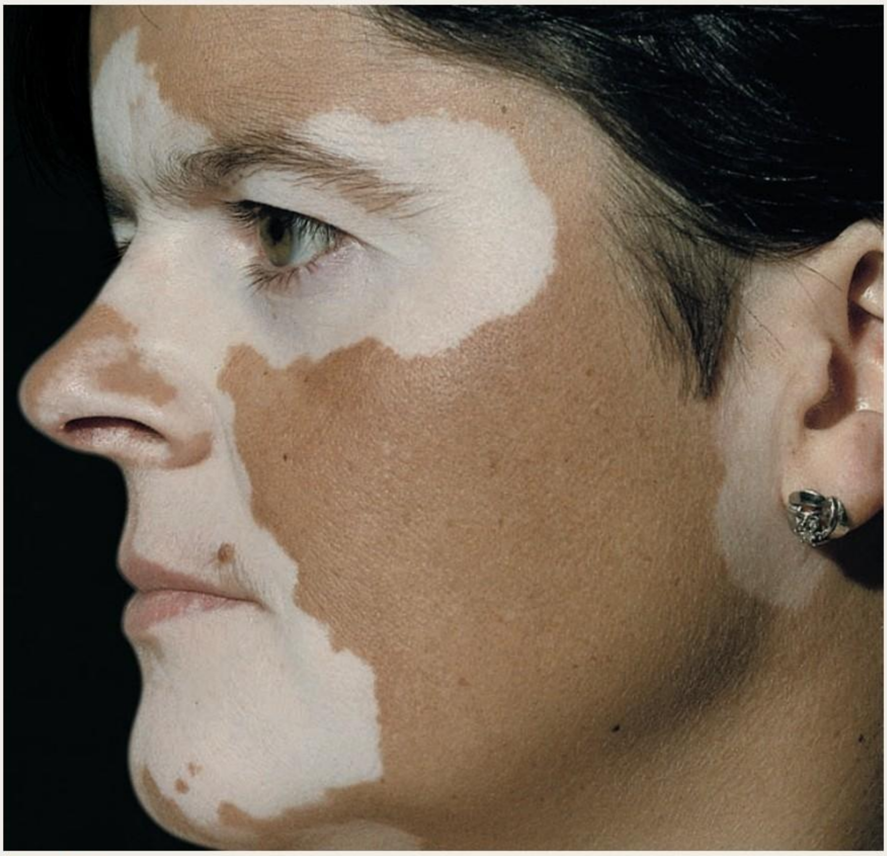


Figure 13-2. Vitiligo: face Extensive depigmentation of the central face. Involved vitiliginous skin has convex borders, extending into the normal pigmented skin. Note the chalk-white color and sharp margination. *Note also that the dermal nevomelanocytic nevus on the upper lip has retained its pigmentation.*

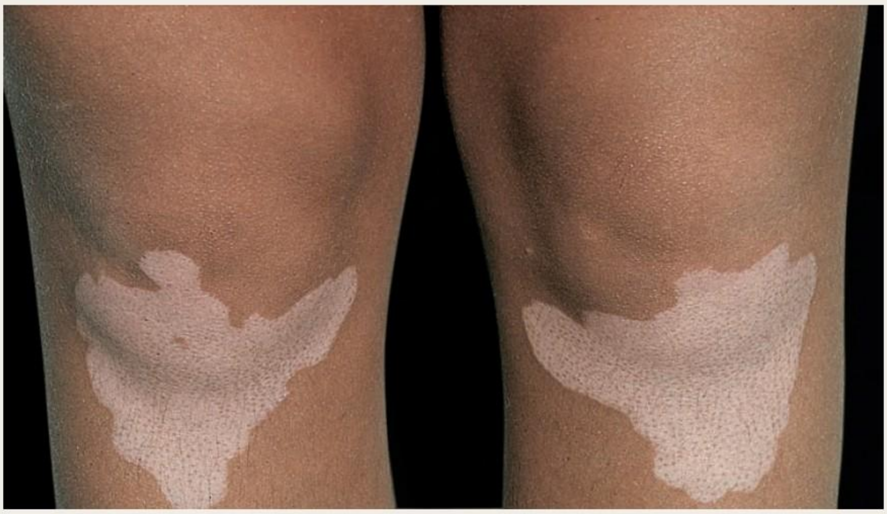


Figure 13-3. Vitiligo: knees Depigmented, sharply demarcated macules on the knees. Apart from the loss of pigment, vitiliginous skin appears normal. There is striking symmetry. Note tiny follicular pigmented spots within the vitiligo areas that represent repigmentation.

Distribution. Two general patterns. The *focal* type is characterized by one or several macules in a single site; this may be an early evolutionary stage of one of the other types in some

photoaging as well as solar keratoses may occur in vitiligo macules in those with history of long exposures to sunlight. Squamous cell carcinoma, limited to the white macules, has present, is very stable. May be associated with vitiligo elsewhere.

Associated Cutaneous Findings. White hair and prematurely gray hair. Circumscribed areas of white hair, analogous to vitiligo macules, are called *poliosis*. Alopecia areata (see Section 33) and halo nevi (see Section 9). In older patients,

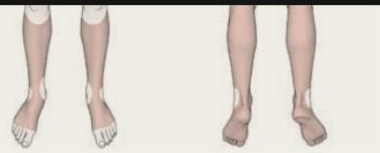


Figure 13-4. Vitiligo: predilection sites.

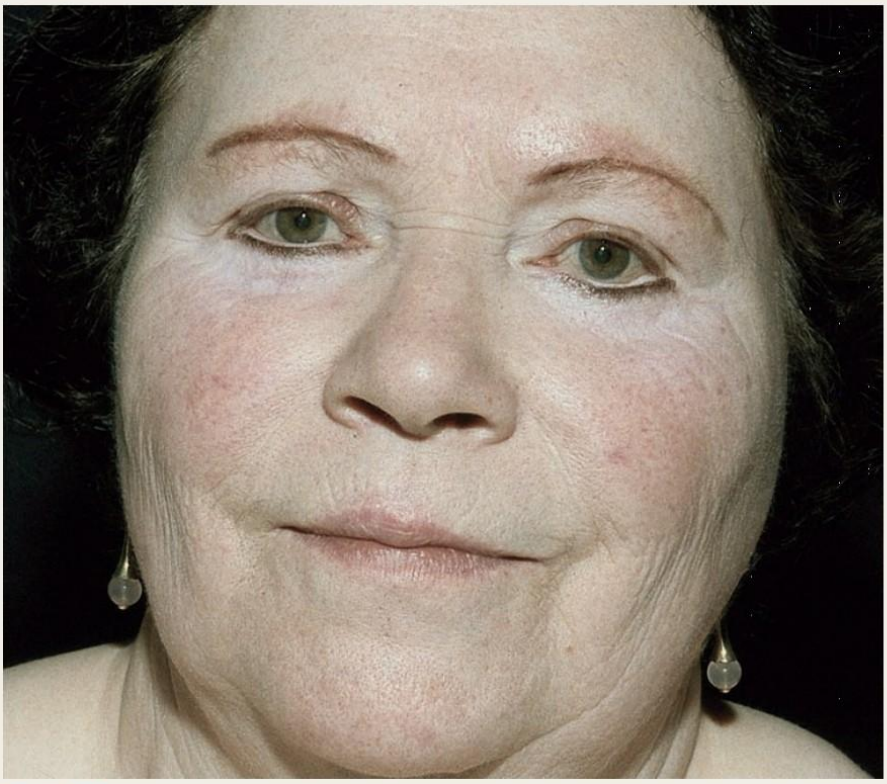


Figure 13-5. Universal vitiligo Vitiliginous macules have coalesced to involve all skin sites with complete depigmentation of skin and hair in a female. The patient is wearing a black wig and has darkened the brows with eyebrow pencil and eyelid margins with eye liner.

General Examination. Rarely associated with thyroid disease, Hashimoto thyroiditis (Graves disease); also diabetes mellitus—probably <5%; pernicious anemia (uncommon, but increased risk); Addison disease (very uncommon); and multiple endocrinopathy syndrome (rare). Ophthalmologic examination may reveal evidence of healed chorioretinitis or iritis. Vision is unaffected. Hearing is normal. The *Vogt-Koyanagi-Harada syndrome* is vitiligo + poliosis + uveitis + dysacusis + alopecia areata.

Laboratory Examinations

Wood Lamp Examination. For identification of vitiligo macules in very light skin.

Dermatopathology. In certain difficult cases, a

show normal skin except for an absence of melanocytes.

Electron Microscopy Absence of melanocytes and of melanosomes in keratinocytes.

Laboratory Studies. Thyroxine (T_4), thyroid-stimulating hormone (radioimmunoassay), fasting blood glucose, complete blood count with indices (pernicious anemia), ACTH stimulation test for Addison disease, if suspected.

Diagnosis

Normally, diagnosis of vitiligo can be made readily on clinical examination of a patient with progressive, acquired, chalk-white, bilateral (usually symmetric), sharply defined mac-

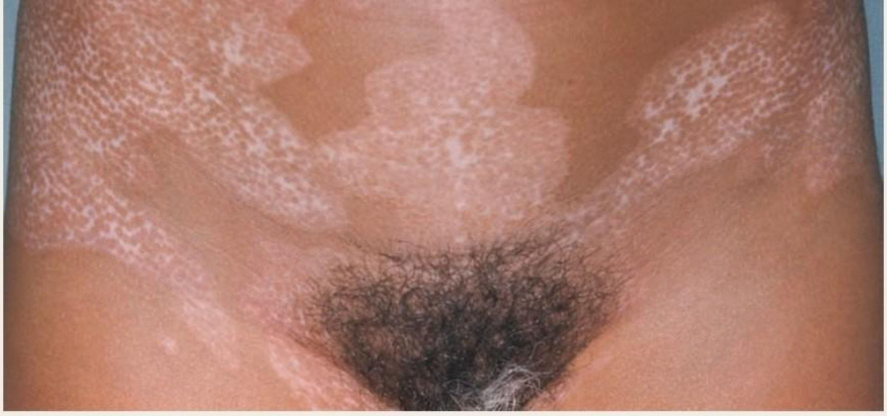


Figure 13-6. Vitiligo repigmentation A follicular pattern of repigmentation due to PUVA therapy occurring in a large vitiliginous macule on the lower abdomen. By confluence of the macules, the vitiliginous areas have almost filled in but are still lighter than the surrounding normal skin. Melanocytes may persist in the hair follicle epithelium and serve to repopulate involved skin, spontaneously or with photochemotherapy.

Note: Response to all treatments is slow. When it occurs, it is signaled by tiny, usually follicular macules of pigmentation (Fig. 13-6).

Minigrafting

Minigrafting (autologous Thiersch grafts, suction blister grafts, autologous mini-punch grafts, transplantation of cultured autologous melanocytes) may be a useful technique for refractory and stable segmental vitiligo macules. "Pebbling" of the grafted site may occur.

Depigmentation

The objective of depigmentation is "one" skin color in patients with extensive vitiligo or in those who have failed or reject other treatments.

Treatments. Bleaching of *normally pigmented skin* with monobenzylether of hydroquinone 20% (MEH) cream is a permanent, irreversible process. The success rate is >90%. The end-stage color of depigmentation with MEH is chalk-white, as in vitiligo macules.

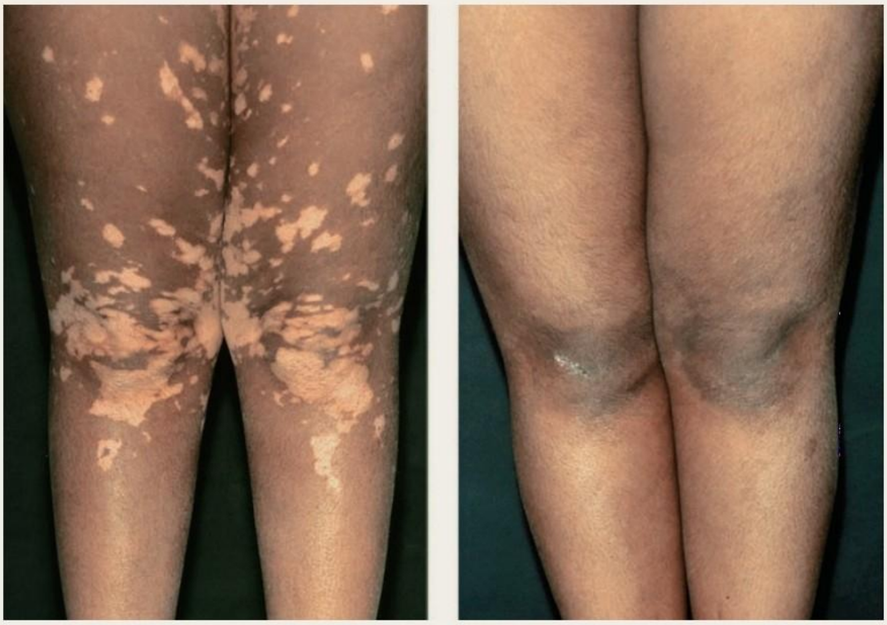


Figure 13-7. Vitiligo: therapy-induced repigmentation This 20-year-old Indian female is being treated with photochemotherapy (PUVA). There is slight erythema in the vitiliginous macules in the early phases (left) of therapy that will be followed by follicular pigmentation as in Fig. 13-6; after 1 year of treatment, vitiligo has completely repigmented, but there is now hyperpigmentation of the knees (right). This, however, will fade with time and the color of the repigmented areas will blend with that of the surrounding skin.

- Skin varied depending on type. "Snow white," creamy white (Fig. 13-8; Table 13-1), light tan.
- Hair: white (tyrosinase negative; Fig. 13-8A); yellow, cream, light brown (tyrosinase positive); red, platinum (Table 13-1).
- Eyes: nystagmus, reduction of visual activity, iris translucency (Fig. 13-8B), decreased retinal pigment, foveal hyperplasia, strabismus.
- Management: No treatment available. Albinos should be under care of an ophthalmologist (vision problems) and a dermatologist (sun protection and detection of skin cancer).
- National volunteer group of albinos [in the United States: NOAH—*N*ational *O*rganization for *A*lbinism and *H*ypomelanosis (Noah of the Old Testament was alleged to be an Albino)].

TABLE 13-1 CLASSIFICATION OF ALBINISM

Type	Subtypes	Gene Locus	Includes	Clinical Findings
OCA1	OCA1A	*TYR*	Tyrosinase-negative OCA	White hair and skin, eyes (pink at birth → blue)
	OCA1B	*TYR*	Minimal pigment OCA	White to near-normal skin and hair pigmentation
			Platinum OCA	
			Yellow OCA	Yellow (pheomelanin) hair, light red or brown hair
			Temperature-sensitive OCA	May have near-normal pigment but not in axilla
			Autosomal-recessive OCA (some)	
OCA2		*P*	Tyrosinase-positive OCA	Yellow hair, skin "creamy" white (Africa)
			Brown OCA	Light brown/tan skin (Africa)
OCA3		*TYRP1*	Autosomal-recessive OCA (some)	
			Rufous OCA	Red and red-brown skin and brown eyes (Africa)
OCA4		*MATP*		
HPS		*HPS*	Hermansky-Pudlak syndrome	Skin/hair as in OCA1A or OCA1B or OCA2, bleeding diathesis (Puerto Rico)
CHS		*LYST*	Chédiak-Higashi syndrome	Silver hair/hypopigmentation/serious medical problems
OA1		*OA1*	X-linked OA	Normal pigmentation of skin and hair

OCA, oculocutaneous albinism; TYR, tyrosinase; P, pink protein; TYRP1, tyrosinase-related protein 1; OA, ocular albinism; MATP, membrane-associated transporter protein; LYST, lysosome trafficking.
Modified from Bahadoran P et al., in Freedberg IM et al. (eds). *Fitzpatrick's Dermatology in General Medicine*, 6th ed. New York, McGraw-Hill, 2003.

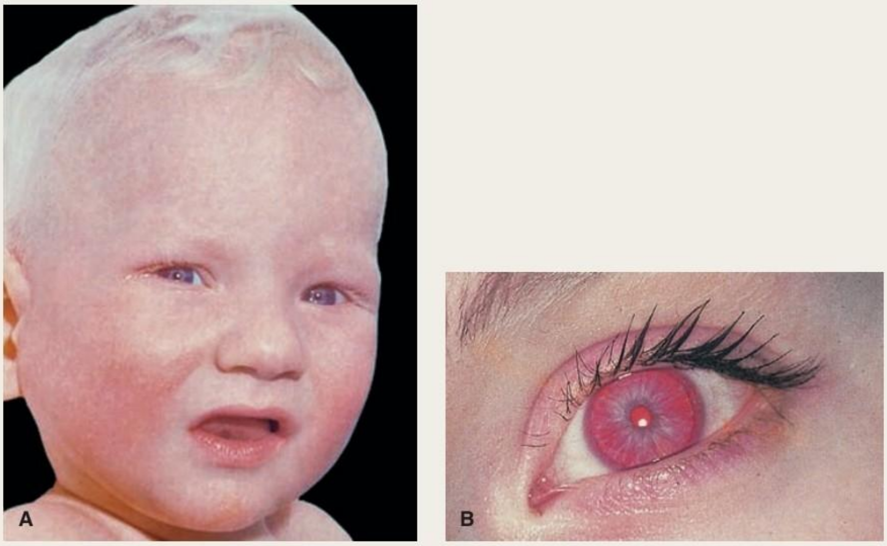


Figure 13-8. **(A) Oculocutaneous albinism** White skin, white eyelashes, eyebrows, and scalp hair. The irises appear translucent. Heme pigment gives the face a pinkish hue. There is squinting due to photophobia and nystagmus. **(B) Iris translucency** is a sine qua non in all types of oculocutaneous albinism, even in those patients in whom the iris is brown. The iris is rarely pink except in infants, and the diagnosis of albinism depends on the detection of iris translucency. This is best done in a dark room with a flashlight pointed at the sclera.

- and living in sunny climates; 10% of patients are men.
- Macular hyperpigmentation mostly sharply defined in the malar and frontal areas of the face (Fig. 13-10). Usually uniform but also blotchy.

development of a disfiguring spotty leukoderma.

- *Prevention: Opaque sun blocks.*
- *Synonyms*: Chloasma (Greek: "a green spot"), mask of pregnancy.

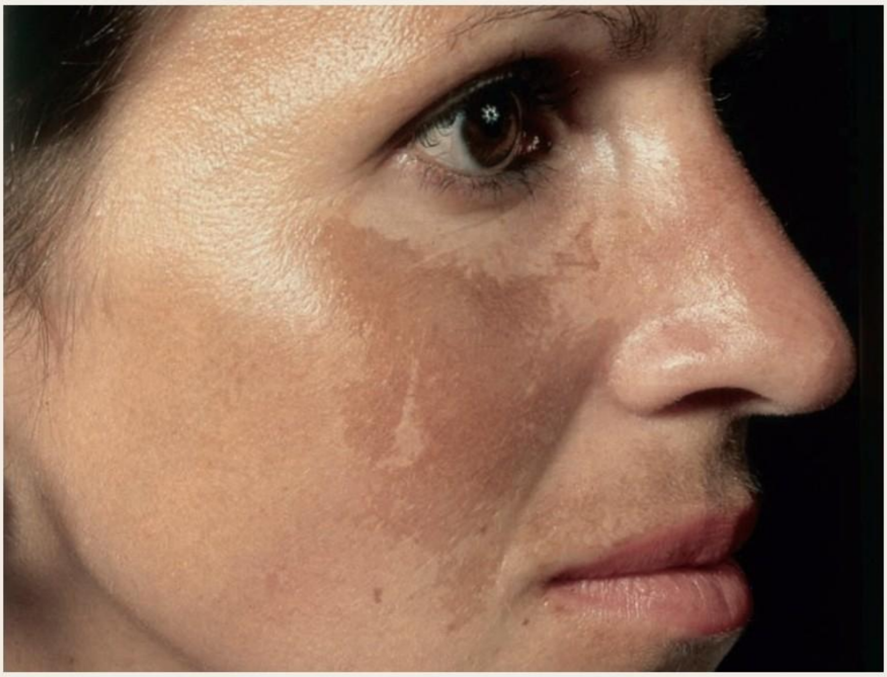


Figure 13-10. **Melasma** Well-demarcated, hyperpigmented macules are seen on the cheek, nose, and upper lip.

Pigmentary Changes Following Inflammation of the Skin

Hyperpigmentation ICD-9: 709.0 ∘ ICD-10: L81.9 ☐ ◐

- *Postinflammatory epidermal melanin hyperpigmentation* is a major problem for patients with skin phototypes IV, V, and VI (Figs. 13-11 and 13-12). This disfiguring pigmentation can develop with acne (Fig. 13-11), psoriasis, lichen planus (Fig. 13-12), atopic dermatitis, or contact dermatitis or after any type of trauma to the skin. It may persist for weeks to months but does respond to topical hydroquinone, which accelerates its disappearance. Lesions are characteristically limited to the site of the preceding inflammation and usually have indistinct, feathered borders.
- Some drug eruptions may be associated with dermal melanin hyperpigmentation (Fig. 13-12), which may also be associated with lichen planus and cutaneous lupus erythematosus. This dermal hyperpigmentation may be persistent, and there is no treatment.
- *Riehl melanosis* (melanodermatitis toxica) is a reticular, confluent black to brown-violet pigmentation of the face and neck (Fig. 13-14). It may be a result of contact sensitivity or photocontact sensitivity related to chemicals, particularly fragrance in cosmetics.

For hypermelanosis due to phototoxic reactions induced by psoralens (Berloque dermatitis), see Section 10, and for *nonmelanin-based hyperpigmentation* due to drugs, see Section 23.

Figure 13-11. **Hypermelanosis with acne** In this 30-year-

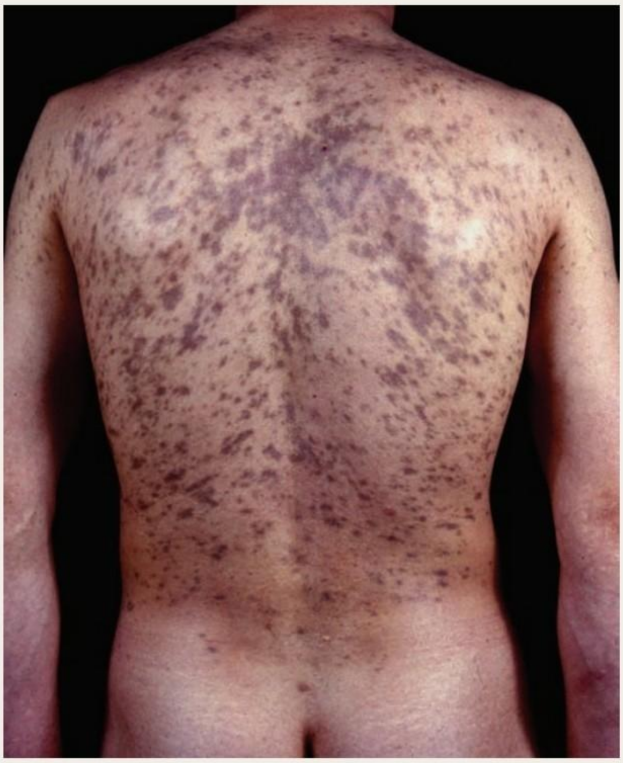


Figure 13-12. **Postinflammatory hyperpigmentation** This may follow a drug eruption, or lichen planus, especially in skin phototypes V and VI, as was the case in this middle-aged East Indian man. There is a condition described as Ashy dermatosis, which is clinically indistinguishable from postinflammatory hyperpigmentation following lichen planus as shown here. Postinflammatory hyperpigmentation is a major problem in young females with skin phototypes IV and V.

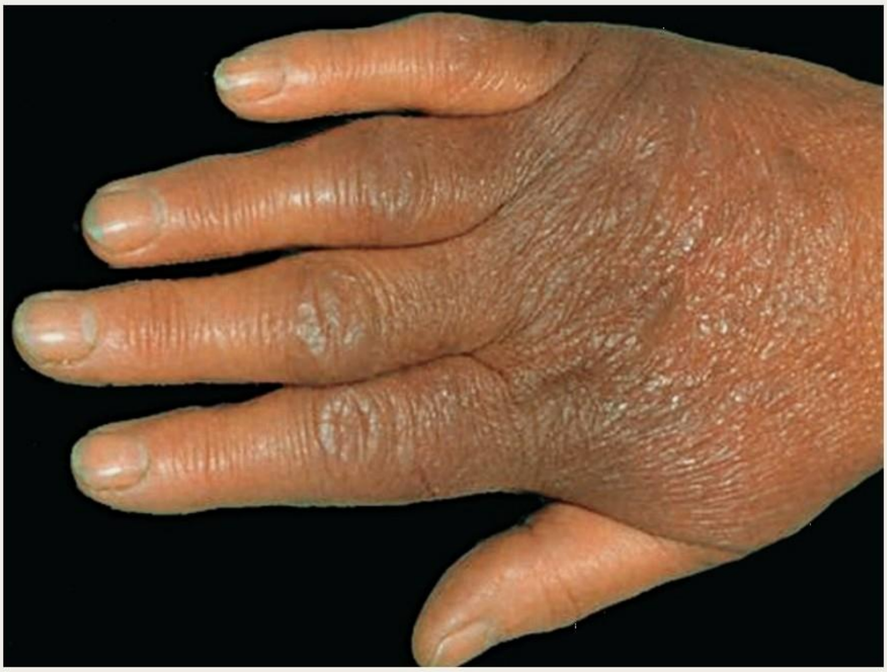


Figure 13-13. **Postinflammatory dermal hyperpigmentation** This is shown on the hand of a skin phototype IV African woman following a fixed drug eruption.

Hypopigmentation ICD-9: 709.0 ◦ ICD-10: L81.9 ☐ ●

- Postinflammatory hypomelanosis is always related to loss of melanin. It is a special feature of pityriasis versicolor (Fig. 13-15, see also Section 26), in which the hypopigmentation may also remain for weeks after the active infection has disappeared.
- Hypomelanosis is not uncommonly seen in atopic dermatitis, psoriasis (Fig. 13-16), guttate parapsoriasis, and pityriasis lichenoides chronica.
- It may also be present in cutaneous lupus erythematosus (Fig. 13-17), alopecia mucinosa, mycosis fungoides, lichen striatus, seborrheic dermatitis, and leprosy.
- Hypomelanosis may follow dermabrasion and chemical peels; in these conditions, there is a "transfer block," in which melanosomes are present in melanocytes but are not transferred to keratinocytes, resulting in hypomelanosis. The lesions are usually not chalk-white, as in vitiligo, but "off" white and have indiscrete margins.
- A common type of hypopigmentation is associated with *pityriasis alba* (Fig. 13-18). This is a macular hypopigmentation mostly on the face of children, off-white with a powdery scale. Relatively indistinct margins under Wood light and scaling distinguish this eczematous dermatitis from vitiligo. It is self-limited.
- Hypomelanosis not uncommonly follows intralesional glucocorticoid injections; but when the injections are stopped, a normal pigmentation develops in the areas.
- Depending on the associated disorder, postinflammatory hypomelanosis may respond to oral PUVA photochemotherapy.

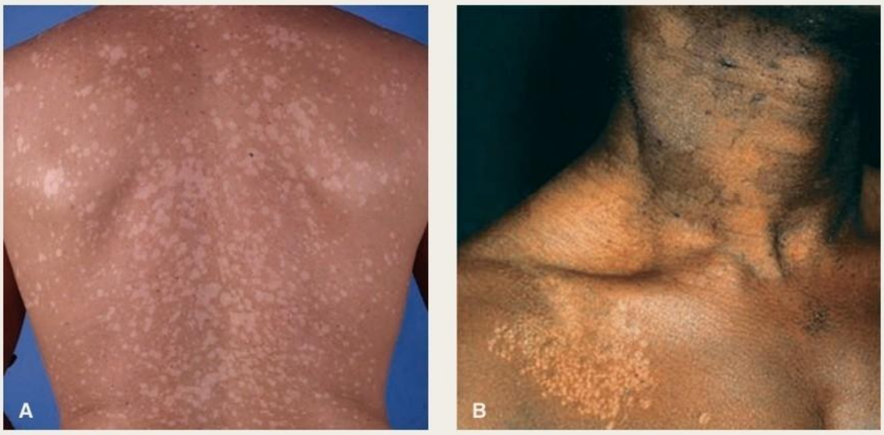


Figure 13-15. Pityriasis versicolor (A) Hypopigmented, sharply marginated, scaling macules on the back of an individual with skin phototype III. Gentle abrasion of the surface will accentuate the scaling. This type of hypomelanosis can remain long after the eruption has been treated and the primary process is resolved. **(B) Pityriasis versicolor in African skin** Lesions are perifollicular on the chest and coalesce to large confluent patches on the neck where the fine scaling can best be seen.

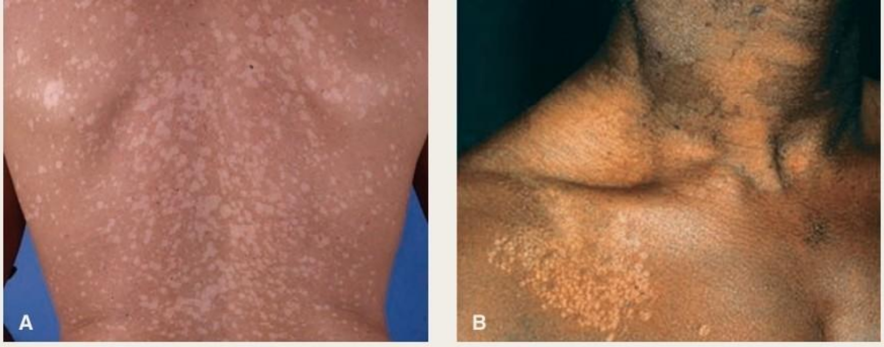


Figure 13-15. Pityriasis versicolor (A) Hypopigmented, sharply marginated, scaling macules on the back of an individual with skin phototype III. Gentle abrasion of the surface will accentuate the scaling. This type of hypomelanosis can remain long after the eruption has been treated and the primary process is resolved. **(B) Pityriasis versicolor in African skin** Lesions are perifollicular on the chest and coalesce to large confluent patches on the neck where the fine scaling can best be seen.

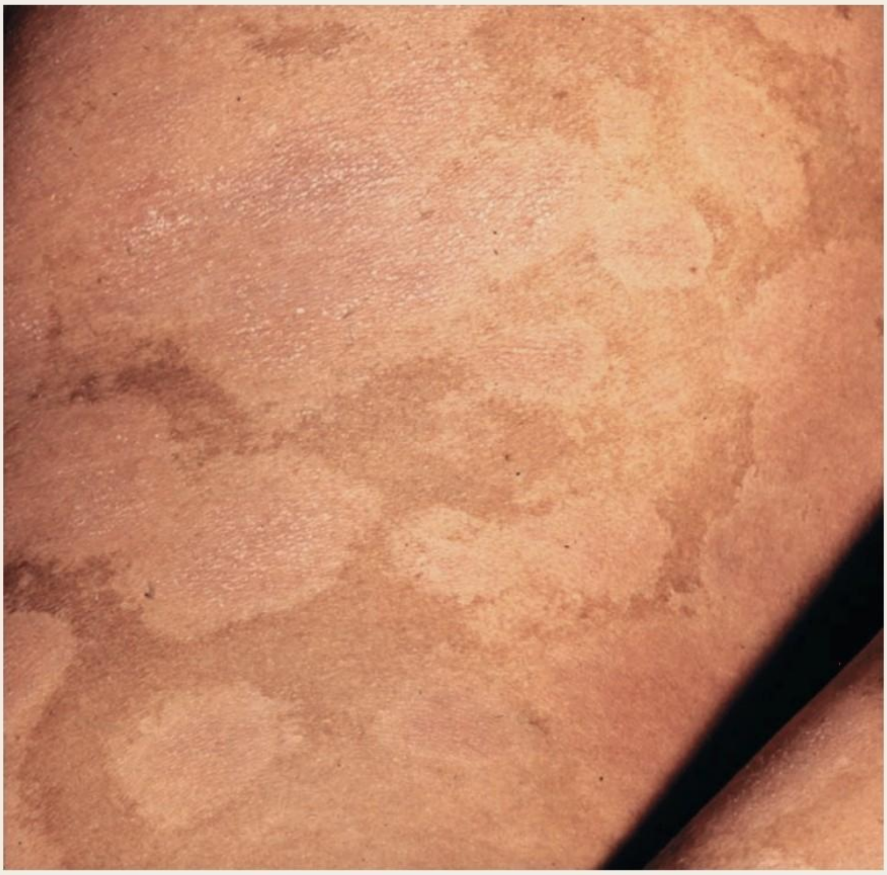


Figure 13-16. Postinflammatory hypomelanosis (psoriasis) The hypomelanotic lesions correspond exactly to the antecedent eruption. There is some residual psoriasis within the lesions.

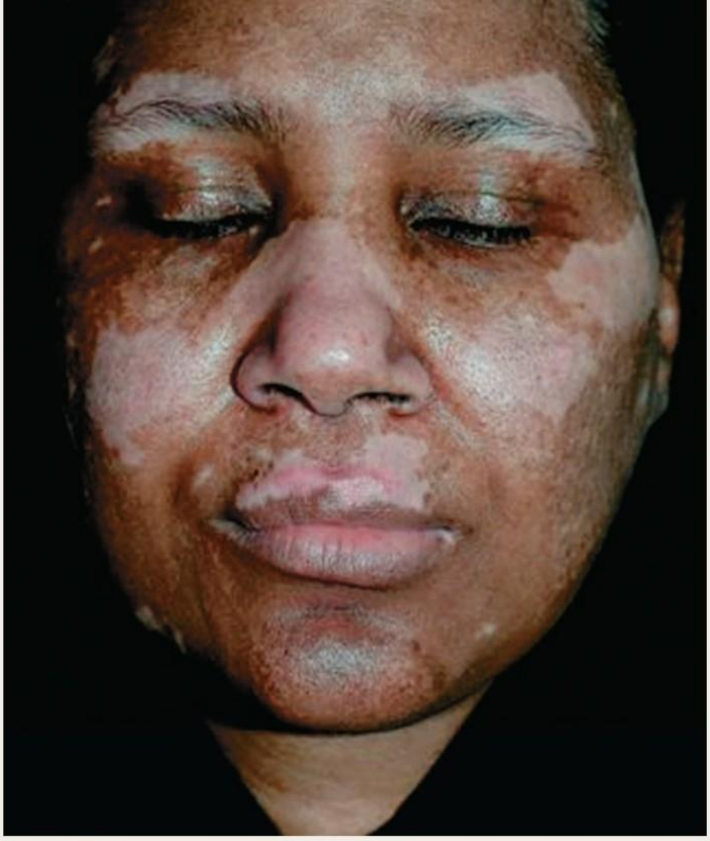


Figure 13-17. Postinflammatory hypopigmentation in a 33-year-old Vietnamese female. The patient had had chronic cutaneous lupus erythematosus. Residual inflammation of lupus is still seen on the upper lip.

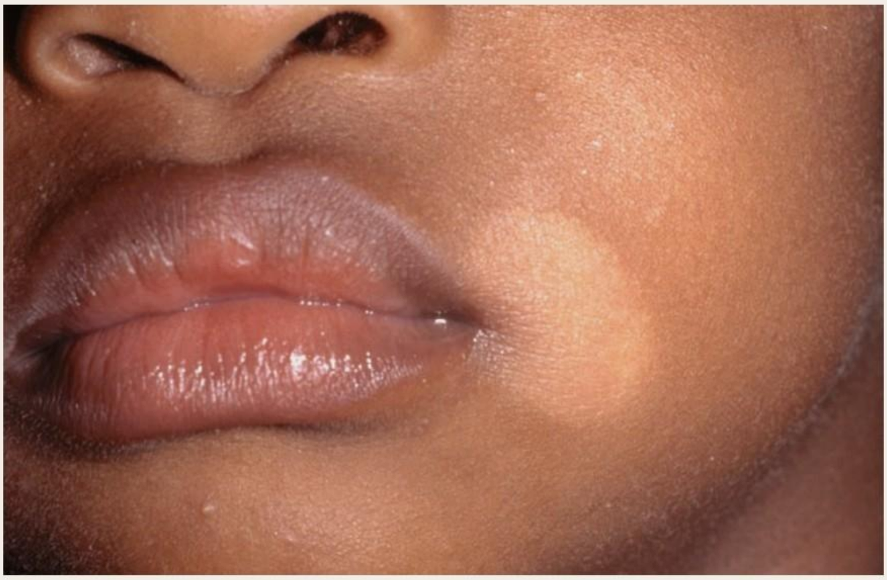


Figure 13-18. Pityriasis alba A common disfiguring hypomelanosis, which, as the name indicates, is a white area (alba) with very mild scaling (pityriasis). It is observed in a large number of children in the summer in temperate climates. It is mostly a cosmetic problem in persons with brown or black skin and commonly occurs on the face, as in this child. Among 200 patients with pityriasis alba, 90% ranged from 6 to 12 years of age. In young adults, PA quite often occurs on the arms and trunk.

- Urticaria is composed of wheals (transient edematous papules and plaques, usually pruritic and due to edema of the papillary body) (Fig. 14-6; also see Fig. 14-8). The wheals are superficial, well defined.
- Angioedema is a larger edematous area that involves the dermis *and* subcutaneous tissue (Fig. 14-7) and is deep and ill defined. Urticaria and angioedema are thus the same edematous process but involving different levels of the cutaneous vascular plexus: papillary and deep.
- Urticaria and/or angioedema may be acute recurrent or chronic recurrent.
- Other forms of urticaria/angioedema are recognized: IgE and IgE receptor dependent, physical, contact, mast cell degranulation related, and idiopathic.
- In addition, angioedema/urticaria can be mediated by bradykinin, the complement system, and other effector mechanisms.
- Urticarial vasculitis is a special form of cutaneous necrotizing venulitis (see p. 363).
- There are some syndromes with angioedema in which urticarial wheals are rarely present (e.g., hereditary angioedema).

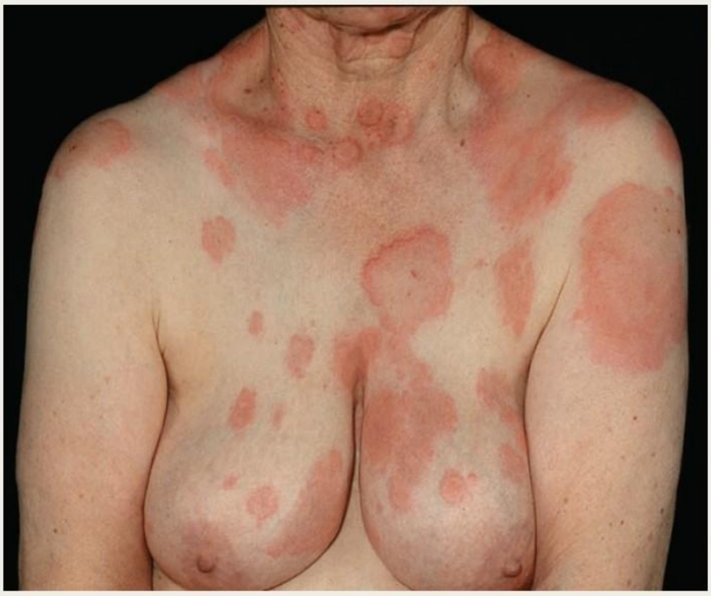


Figure 14-6. Acute urticaria Small and large wheals with erythematous borders and a lighter color centrally. Well defined. The lesion on the left upper arm is ill defined at its lower border where it is regressing.

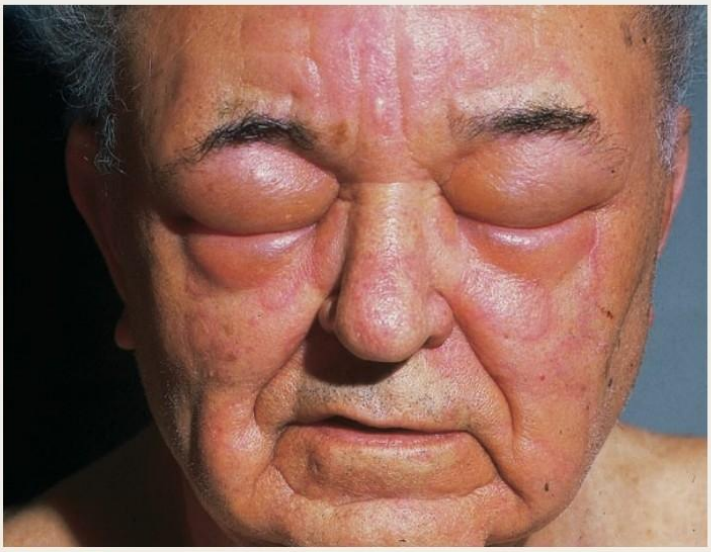


Figure 14-7. Acute urticaria and angioedema Note that there are both superficial wheals and deep, diffuse edema. Occurred after the patient had eaten shellfish. He had similar episodes previously but never considered seafood as the cause.

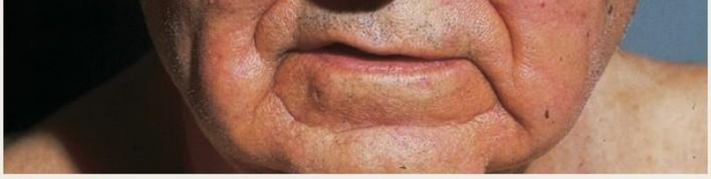


Figure 14-7. Acute urticaria and angioedema Note that there are both superficial wheals and deep, diffuse edema. Occurred after the patient had eaten shellfish. He had similar episodes previously but never considered seafood as the cause.

Epidemiology and Etiology

Incidence. 15–23% of the population may have had this condition during their lifetime.

Etiology. Urticaria/angioedema is not a disease but a cutaneous reaction pattern. For classification and etiology, see Table 14-1.

Clinical Types

Acute Urticaria. Acute onset and recurring over <30 days. Usually large wheals often associated with angioedema (Figs. 14-6 and 14-7); often IgE dependent with atopic diathesis; related to foods, parasites, and penicillin. Also, complement mediated in serum sickness–like reactions (whole blood, immunoglobulins, penicillin). Often accompanied by angioedema. Common. (See also “Drug-Induced Acute Urticaria” in Section 23.)

Chronic Urticaria. Recurring over <30 days. Small and large wheals (Fig. 14-8). Rarely IgE dependent but often due to anti-FcεR autoantibodies; etiology unknown in 80% and therefore considered idiopathic. Intolerance to salicylates, benzoates. Common. Chronic urticaria affects adults predominantly and is approximately twice as common in women as in men. Up to 40% of patients with chronic urticaria of >6 months’ duration still have urticaria 10 years later.

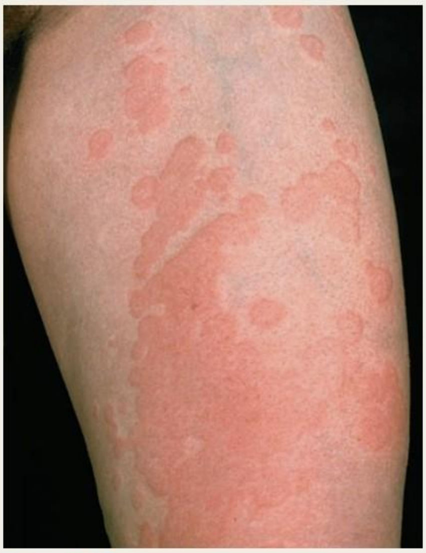


Figure 14-8. Chronic urticaria Chronic urticaria of 5-year duration in an otherwise healthy 35-year-old female. Eruptions occur on an almost daily basis and, as they are highly pruritic, greatly impair the patient’s quality of life. Although suppressed by antihistamines, there is an immediate recurrence after treatment is stopped. Repeated laboratory and clinical examinations have not revealed an apparent cause.

TABLE 14-1 ETIOLOGY AND CLASSIFICATION OF URTICARIA/ANGIOEDEMA

- Immunologic
  - IgE-mediated urticaria
  - Complement-mediated urticaria
  - Autoimmune urticaria
  - Immune contact urticaria
- Physical
  - Dermographism
  - Cold urticaria
  - Solar urticaria
  - Cholinergic urticaria
  - Pressure angioedema
  - Vibratory angioedema
- Urticaria due to mast cell–releasing agents, pseudoallergens, ACE inhibitors
- Idiopathic urticaria
- Nonimmune contact urticaria
- Urticaria associated with vascular/connective tissue autoimmune disease
- Distinct angioedema (± urticaria) syndromes
  - Hereditary angioedema
  - Angioedema–urticaria–eosinophilia syndrome

Symptoms. Pruritus. In angioedema of palms and soles pain. Angioedema of tongue, pharynx interferes with speech, food intake, and breathing. Angioedema of larynx may lead to asphyxia.

Clinical Manifestation

Skin Lesions. Sharply defined *wheals* (Fig. 14-6), small (<1 cm) to large (>8 cm), erythematous or white with an erythematous rim, round, oval, acriform, annular, serpiginous (Figs. 14-6 and 14-8), due to confluence and resolution in one area and progression in another (Fig. 14-8). Lesions are pruritic and transient.

Angioedema—skin colored, transient enlargement of portion of face (eyelids, lips, tongue) (Figs. 14-7 and 23-5), extremity, or other sites due to subcutaneous edema.

Distribution. Usually regional or generalized.

ing anaphylatoxins that induce mast cell of the trigger mechanism (see below).

Special Features/As Related to Pathogenesis

Immunologic Urticaria. IgE Mediated. Lesions in acute IgE-mediated urticaria result from antigen-induced release of biologically active molecules from mast cells or basophilic leukocytes sensitized with specific IgE antibodies (type I anaphylactic hypersensitivity). Released mediators increase venular permeability and modulate the release of biologically active molecules from other cell types. Often with atopic background. Antigens: food (milk, eggs, wheat, shellfish, nuts), therapeutic agents, drugs (penicillin) (see also "Drug-Induced Acute Urticaria, Angioedema, Edema, and Anaphylaxis" in Section 23), helminths. Most often acute (Figs. 14-6 and 23-5).

Complement Mediated. *Acute.* By way of immune complexes activating complement and releas-

Autoimmune. Common, chronic. Autoantibodies against FcεRI and/or IgE. Positive autologous serum skin test. Clinically, patients with these autoantibodies (up to 40% of patients with chronic urticaria) are indistinguishable from those without them (Fig. 14-8). These autoantibodies may explain why plasmapheresis, intravenous immunoglobulins, and cyclosporine induce remission of disease activity in these patients.

Immunologic Contact Urticaria. Usually in children with atopic dermatitis sensitized to environmental allergens (grass, animals) or individuals sensitized to wearing latex rubber gloves; can be accompanied by anaphylaxis.

Physical Urticarias. Dermographism. Linear urticarial lesions occur after stroking or scratching the skin; they itch and fade in 30 min (Fig. 14-9); 4.2% of the normal population have it; symptomatic dermographism is a nuisance.

Cold Urticaria. Usually in children or young adults; urticarial lesions confined to sites exposed to cold occurring within minutes after rewarming. "Ice cube" test (application of an ice cube for a few minutes to skin) causes wheal.

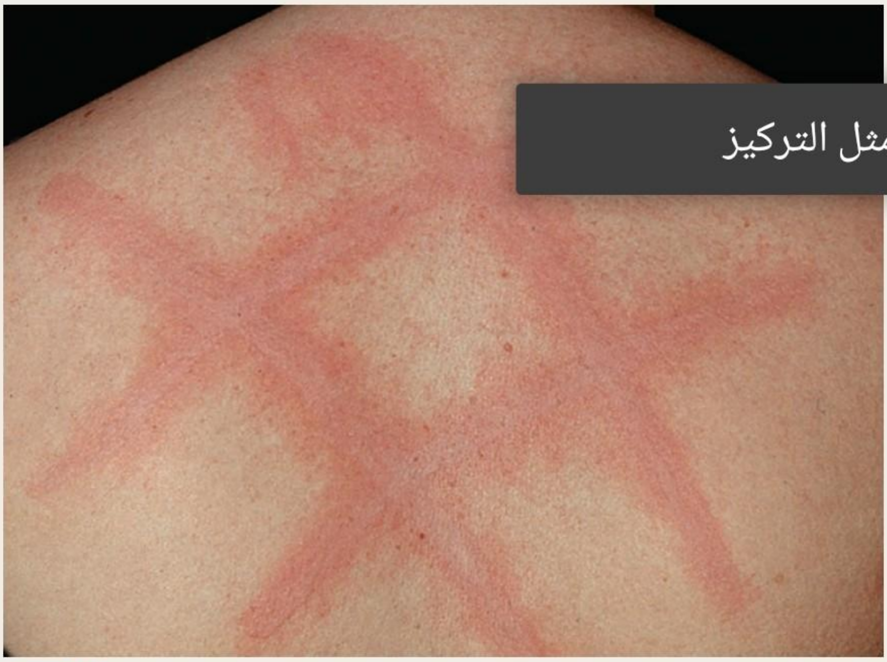


Figure 14-9. Urticaria: dermographism Urticaria as it appeared 5 min after the patient was scratched on the back. The patient had experienced generalized pruritus for several months with no spontaneously occurring urticaria.

Solar Urticaria. Urticaria after solar exposure. Action spectrum from 290 to 500 nm; whealing lasts for <1 h, may be accompanied by syncope; histamine is one of the mediators (see Section 10 and Fig. 10-11).

Cholinergic Urticaria. Exercise to the point of sweating provokes typical small, papular, highly pruritic urticarial lesions (Fig. 14-10). May be accompanied by wheezing.

Aquagenic Urticaria. Very rare. Contact with water of any temperature induces eruption similar to cholinergic urticaria.

Pressure Angioedema. Erythematous swelling induced by sustained pressure (buttock swelling when seated, hand swelling after hammering, foot swelling after walking). Delayed (30 min to 12 h). Painful, may persist for several days, and interferes with quality of life. No laboratory abnormalities; fever may occur.

Vibration Angioedema. May be familial (autosomal dominant) or sporadic. Rare. It is believed to result from histamine release from mast cells caused by a "vibrating" stimulus—rubbing a towel across the back produces lesions, but direct pressure (without movements) does not.

Urticaria Due to Mast Cell–Releasing Agents and Pseudoallergens and Chronic Idiopathic Urticaria. Urticaria/angioedema and even anaphylaxis-like symptoms may occur with radiocontrast media and as a consequence of intolerance to salicylates, food preservatives and additives (e.g., benzoic acid and sodium benzoate), several azo dyes, including tartrazine and sunset yellow (pseudoallergens) (Fig. 14-8); also to ACE inhibitors. May be acute and chronic. In chronic idiopathic urticaria, histamine derived from mast cells in the skin is considered the major mediator, also eicosanoids and neuropeptides.

Nonimmune Contact Urticaria. Due to direct effects of exogenous urticants penetrating into skin or blood vessels. Localized to site of contact. Sorbic acid, benzoic acid in eye solutions wheal.

Solar Urticaria. Urticaria after solar exposure. Action spectrum from 290 to 500 nm; whealing lasts for <1 h, may be accompanied by syncope; histamine is one of the mediators (see Section 10 and Fig. 10-11).

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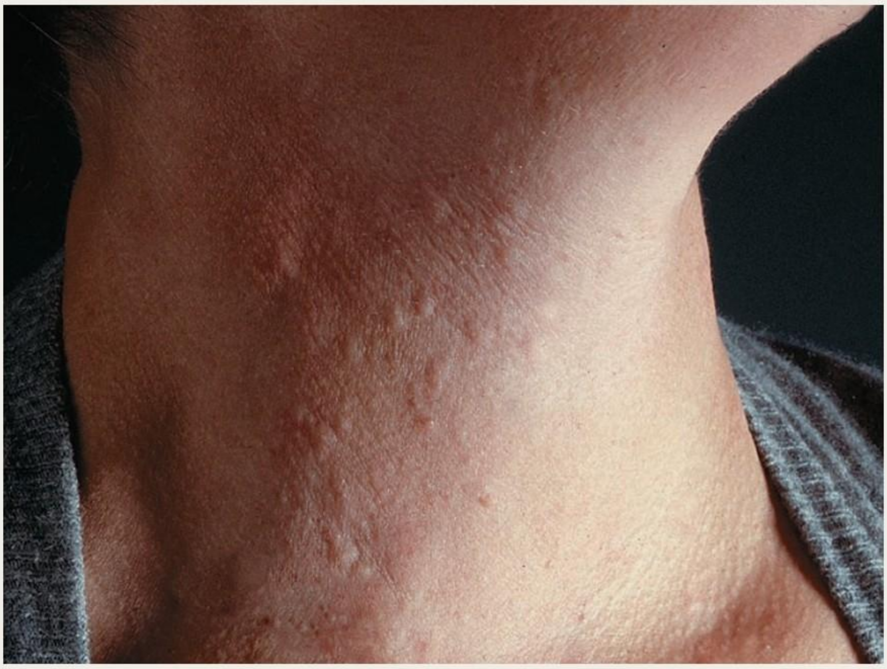


Figure 14-10. Cholinergic urticaria Small urticarial papules on neck occurring within 30 min of vigorous exercise. Papular urticarial lesions are best seen under side lighting.

and foods, cinnamic aldehydes in cosmetics, histamine, acetylcholine, serotonin in nettle stings.

Urticaria Associated with Vascular/Connective Tissue Autoimmune Disease. Urticarial lesions may be associated with systemic lupus erythematosus (SLE) and Sjögren syndrome. However, in most instances, they represent urticarial vasculitis (see p. 363).

Distinct Angioedema (± Urticaria) Syndromes. Hereditary Angioedema (HAE). A serious autosomal-dominant disorder; may follow trauma (physical and emotional). Angioedema of the face (Fig. 14-11) and extremities, episodes of laryngeal edema, and acute abdominal pain caused by angioedema of the bowel wall presenting as surgical emergency. Urticaria rarely occurs. Laboratory abnormalities involve the complement system: decreased levels of C1-esterase inhibitor (85%) or dysfunctional inhibitor (15%), low C4 value in the presence of normal C1 and C3 levels. Angioedema results from bradykinin formation, since C1-esterase inhibitor is also the major inhibitor of the Hageman factor and kallikrein, the two enzymes required for kinin formation.

urticaria, involving the face, neck, extremities, and trunk that lasts for 7–10 days. There is fever and marked increase in normal weight (increased by 10–18%) owing to fluid retention. No other organs are involved. Laboratory abnormalities include striking leukocytosis (20,000–70,000/μL) and eosinophilia (60–80% eosinophils), which are related to the severity of attack. There is no family history. This condition is rare, prognosis is good.

Laboratory Examinations

Serology. Search for hepatitis B–associated antigen, assessment of the complement system, assessment of specific IgE antibodies by radioallergosorbent test (RAST), anti-FcεRI autoantibodies. Serology for lupus and Sjögren syndrome. Autologous serum skin test for autoimmune urticaria.

Hematology. The erythrocyte sedimentation rate (ESR) is often elevated in urticarial vasculitis, and there may be hypocomplementemia; transient eosinophilia in urticaria from reactions to foods, parasites, and drugs; high levels of eosinophilia in the angioedema–urticaria–

trauma (physical and emotional). Angioedema of the face (Fig. 14-11) and extremities, episodes of laryngeal edema, and acute abdominal pain caused by angioedema of the bowel wall presenting as surgical emergency. Urticaria rarely occurs. Laboratory abnormalities involve the complement system: decreased levels of C1-esterase inhibitor (85%) or dysfunctional inhibitor (15%), low C4 value in the presence of normal C1 and C3 levels. Angioedema results from bradykinin formation, since C1-esterase inhibitor is also the major inhibitor of the Hageman factor and kallikrein, the two enzymes required for kinin formation. Episodes can be life threatening.

Angioedema–Urticaria–Eosinophilia Syndrome. Severe angioedema, only occasionally with pruritic

Serology. Search for hepatitis B–associated antigen, assessment of the complement system, assessment of specific IgE antibodies by radioallergosorbent test (RAST), anti-FcεRI autoantibodies. Serology for lupus and Sjögren syndrome. Autologous serum skin test for autoimmune urticaria.

Hematology. The erythrocyte sedimentation rate (ESR) is often elevated in urticarial vasculitis, and there may be hypocomplementemia; transient eosinophilia in urticaria from reactions to foods, parasites, and drugs; high levels of eosinophilia in the angioedema–urticaria–eosinophilia syndrome.

Complement Studies. Screening for functional C1 inhibitor in HAE.

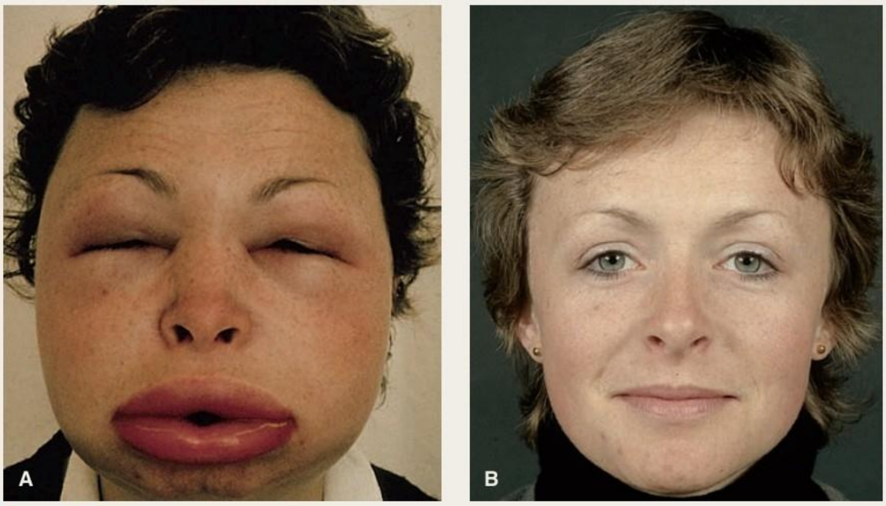


Figure 14-11. Hereditary angioedema (A) Severe edema of the face during an episode leading to grotesque disfigurement. **(B)** Angioedema will subside within hours. These are the normal features of the patient. The patient had a positive family history and had multiple similar episodes including colicky abdominal pain.

Ultrasonography. For early diagnosis of bowel involvement in HAE; if abdominal pain is present, this may indicate edema of the bowel.

Parasitology. Stool specimen for presence of parasites.

Diagnosis

A detailed history (previous diseases, drugs, foods, parasites, physical exertion, solar exposure) is of utmost importance. History should differentiate between *type of lesions*—urticaria, angioedema, or urticaria + angioedema; *duration of lesions* (<1 h or ≥1 h), *pruritus; pain* on walking (in foot involvement), *flushing, burning,* and *wheezing* (in cholinergic urticaria). *Fever* in serum sickness and in the angioedema–urticaria–eosinophilia syndrome; in angioedema, *hoarseness, stridor, dyspnea. Arthralgia* (serum sickness, urticarial vasculitis), *abdominal colicky pain* in HAE. A careful history of medications including penicillin, aspirin, nonsteroidal anti-inflammatory drugs, and ACE inhibitors should be obtained.

Dermographism is evoked by stroking the skin; pressure urticaria is tested by application of pressure (weight) perpendicular to the skin; vibration angioedema by a vibratory stimulus, like rubbing the back with a towel. *Cholinergic urticaria* can best be diagnosed by exercise to sweating and intracutaneous injection of acetylcholine or mecholyl, which will produce micropapular whealing. *Solar urticaria* is verified by testing with UVB, UVA, and visible light (see Fig. 10-11). *Cold urticaria* is verified by a wheal

family history and is characterized by angioedema as the result of trauma, abdominal pain, and decreased levels of C4 and C1-esterase inhibitor.

A practical approach to the diagnosis of urticaria/angioedema is shown in Fig. 14-12 and to angioedema alone in Fig. 14-13.

Course and Prognosis

Half of the patients with urticaria alone are free of lesions in 1 year, but 20% have lesions for >20 years. Prognosis is good in most syndromes except HAE, which may be fatal if untreated.

Management

Prevention by elimination of etiologic chemicals or drugs: aspirin and food additives, especially in chronic recurrent urticaria—rarely successful; prevent trigger in physical urticarias.

Antihistamines. H_1-blockers, e.g., hydroxyzine, terfenadine; or loratadine, cetirizine, fexofenadine; 180 mg/d of fexofenadine or 10–20 mg/d of loratadine usually controls most cases of chronic urticaria, but cessation of therapy usually results in a recurrence; if they fail, H_1 and H_2 blockers (cimetidine) and/or mast cell–stabilizing agents (ketotifen). Doxepin, a tricyclic antidepressant with marked H_1 antihistaminic activity, is valuable when severe urticaria is associated with anxiety and depression.

Prednisone. In *acute* urticaria with angioedema; also for angioedema–urticaria–eosinophilia

Evolution of lesions over several days. May have history of prior EM. May be pruritic or painful, particularly mouth lesions. In severe forms constitutional symptoms such as fever, weakness, malaise.

Skin Lesions. Lesions may develop over ≥10 days. Macule → papule (1–2 cm) → vesicles and bullae in the center of the papule. Dull red. *Iris* or *target-like lesions* result and are typical (Figs. 14-14 and 14-15). Localized to hands and face or

or systemic symptoms. Eruption usually confined to extremities, face, classic target lesions (Figs. 14-14 and 14-15). Recurrent EM minor is usually associated with an outbreak of herpes simplex preceding it by several days.

Severe Forms (EM Major). Most often occurs as a drug reaction, always with mucous membrane involvement; severe, extensive, tendency to become confluent and bullous, positive Nikolsky sign in erythematous lesions (Figs. 14-16 and 14-17). Systemic symptoms: fever,

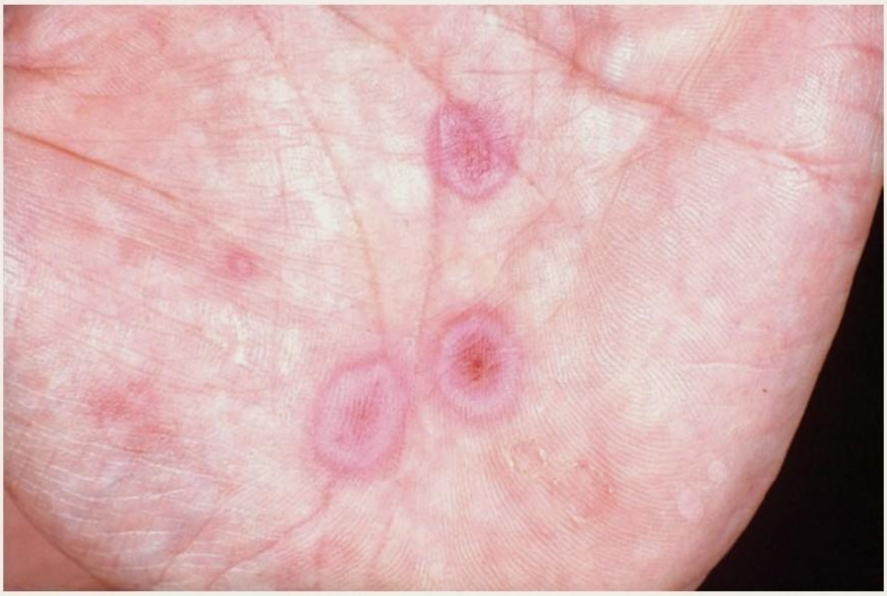


Figure 14-14. Erythema multiforme Iris or target lesions on the palm of a 16-year-old. The lesions are very flat papules with a red rim, a violaceous ring, and a red center.

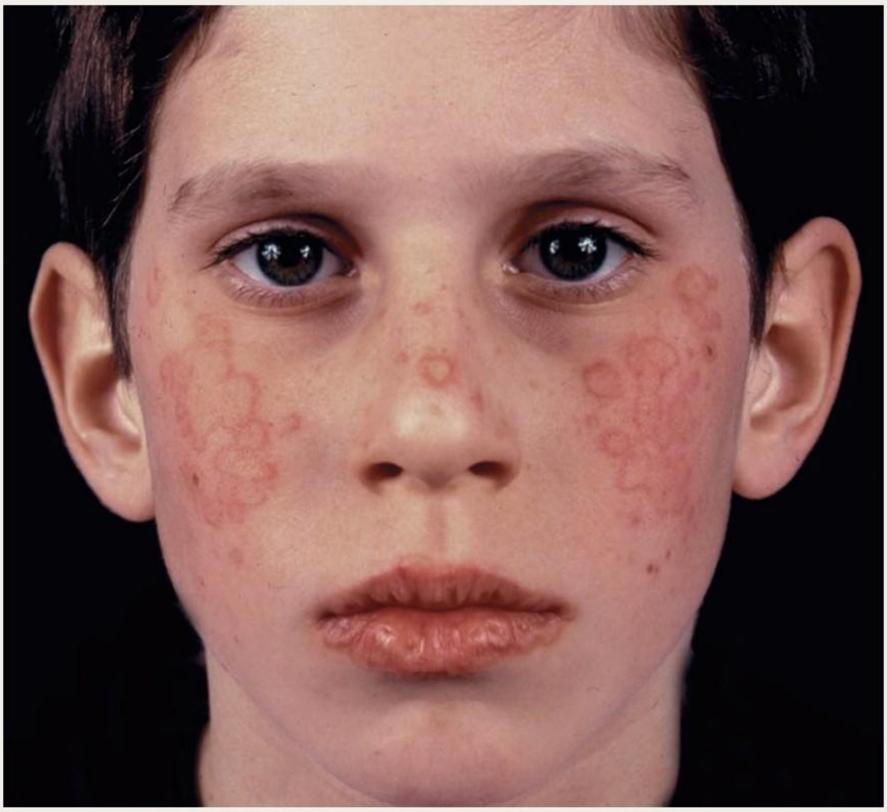


Figure 14-15. Erythema multiforme: minor Multiple, confluent, target-like papules on the face of a 12-year-old boy. The target morphology of the lesions is best seen on the lips.

prostration. Cheilitis and stomatitis interfere with eating; vulvitis and balanitis with mictu-

Acute Exanthematic Eruptions. Drug eruption, psoriasis, secondary syphilis, urticaria, gener-

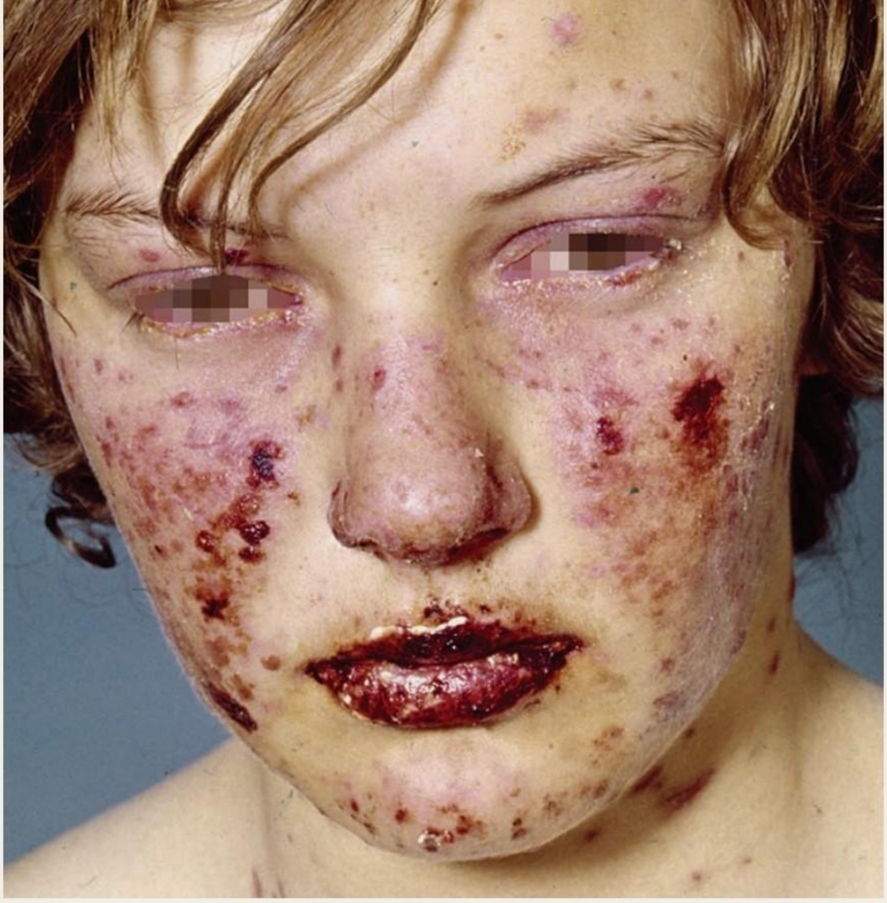


Figure 14-16. Erythema multiforme: major Erythematous, confluent, target-like papules, erosions and crusts on the face. There is erosive and crusted cheilitis indicating mucosal involvement, and there is conjunctivitis. The patient also had a generalized rash consisting of iris lesions.

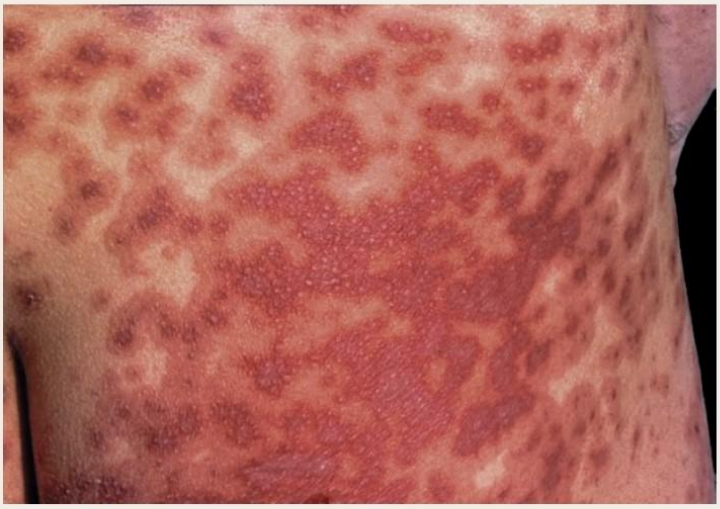


Figure 14-17. Erythema multiforme: major Multiple, target lesions have coalesced, and erosions will develop. This patient had fever and mucosal involvement of mouth, conjunctiva, and genitalia.

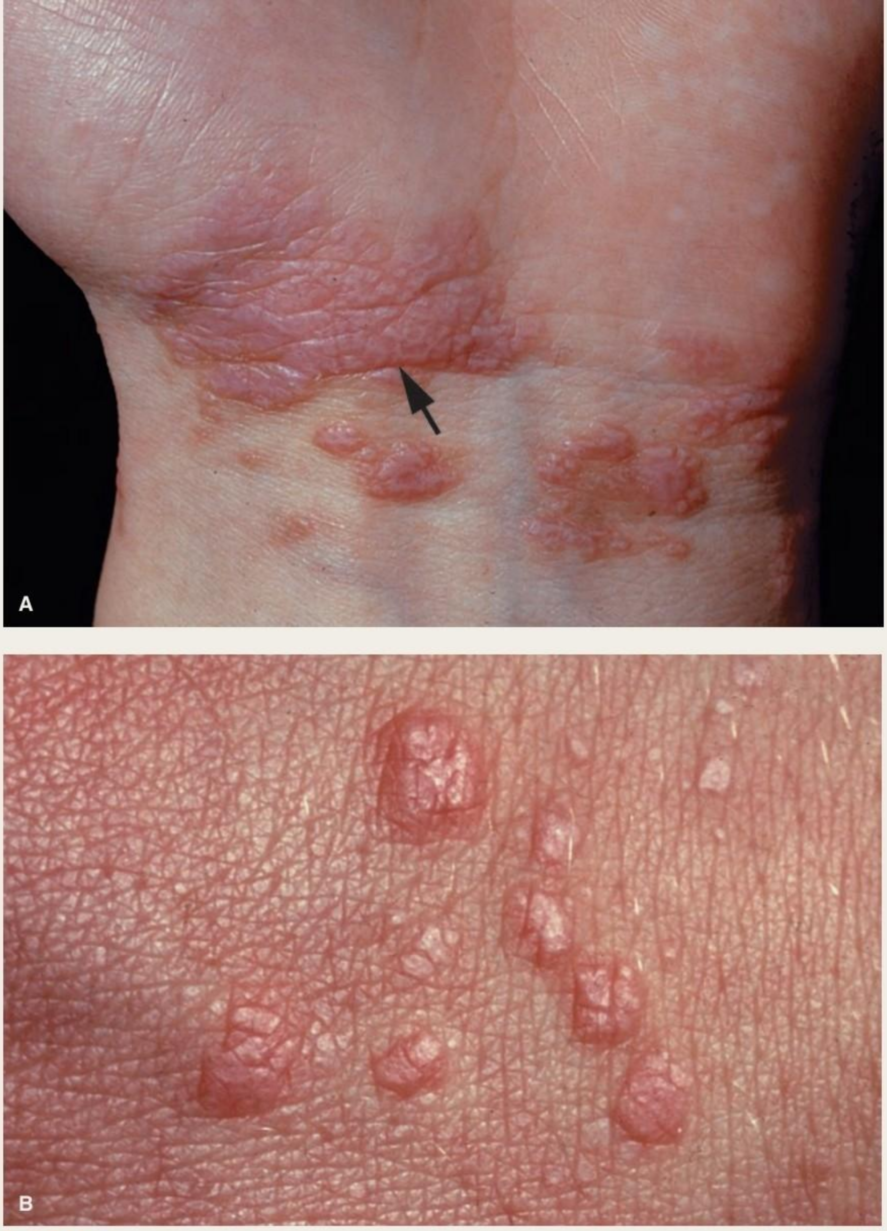


Figure 14-20. Lichen planus (A) Flat-topped, polygonal, sharply defined papules of violaceous color, grouped and confluent. Surface is shiny and, upon close inspection with a hand lens, fine white lines are revealed (Wickham striae, *arrow*). **(B)** Close up of flat-topped shiny violaceous papules that are polygonal.

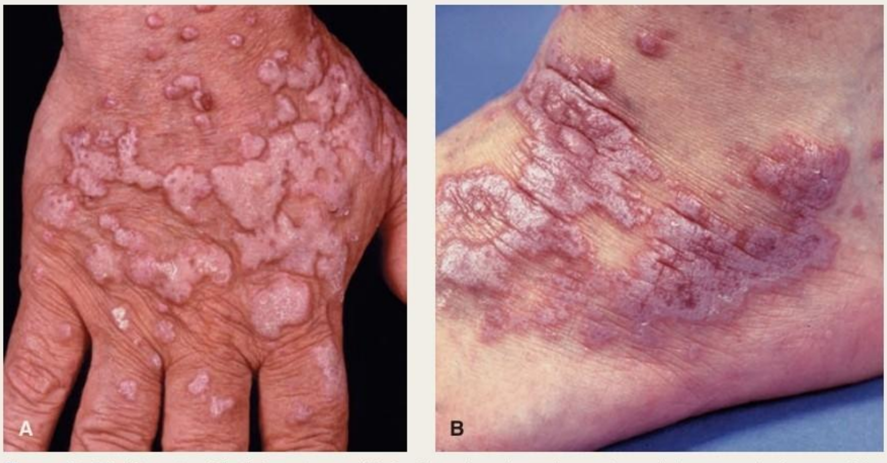


Figure 14-21. Hypertrophic lichen planus (A) Confluent hyperkeratotic papules and plaques on the dorsum of the hand of a light-colored man of African descent. Hyperkeratosis covers Wickham striae, and the characteristic violaceous color of the lesions can be seen only at the very margins. **(B)** Hypertrophic lichen planus on the dorsum of the foot. Lesions form thick plaques with a hyperkeratotic surface and a violaceous border.

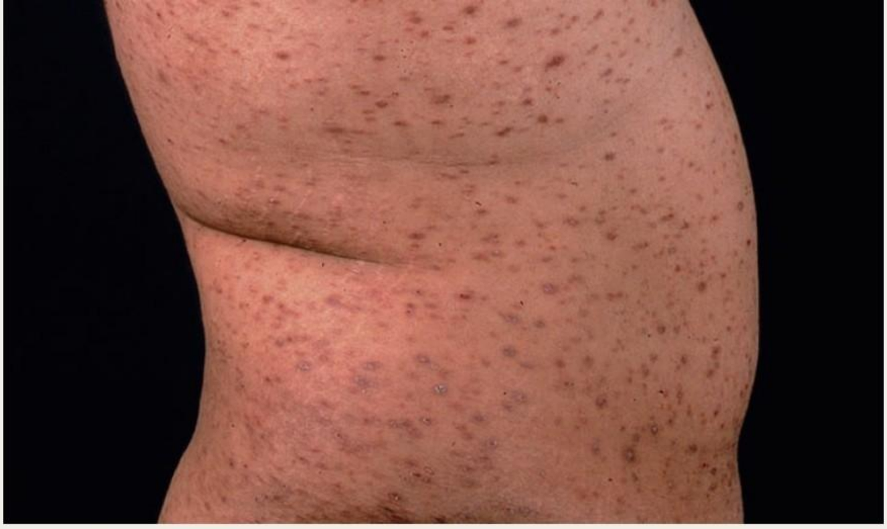


Figure 14-22. Disseminated lichen planus A shower of disseminated papules on the trunk and the extremities (not shown) in a 45-year-old Filipino. Due to the ethnic color of the skin, the papules are not as violaceous as in Caucasians but have a brownish hue.

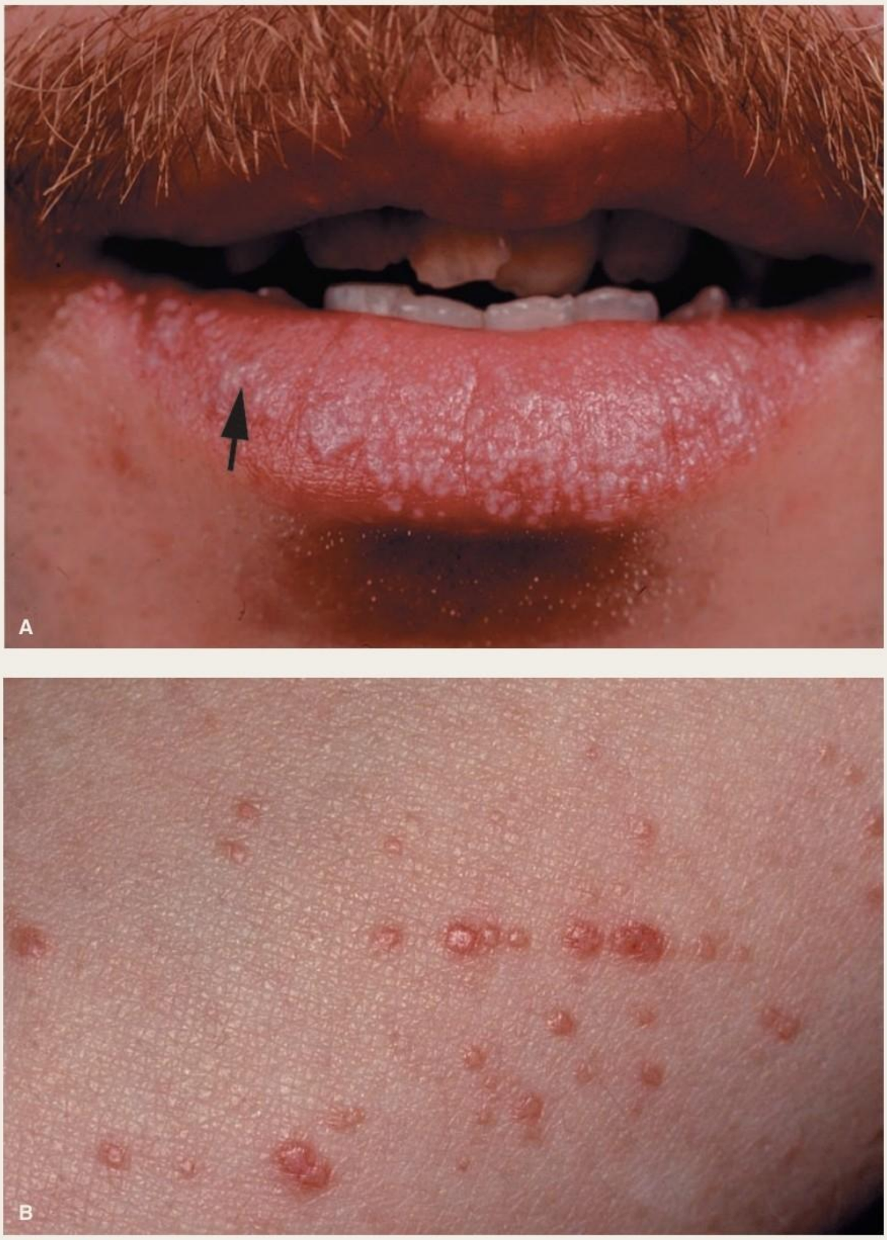


Figure 14-23. Lichen planus (A) Silvery-white, confluent, flat-topped papules on the lips. *Note*: Wickham striae (*arrow*). **(B)** Lichen planus, Koebner phenomenon. Linear arrangement of flat-topped, shiny papules that erupted after scratching.

sions (acute flares) and crusts.

Generalized. Erythematous, discrete, papular, or urticarial lesions on the face, on the dorsa of hands (Fig. 14-34A), arms, and V of the neck.

Others. *Bullae*, often hemorrhagic (acute flares). *Papules* and *scaly plaques* as in SCLE (see Fig. 14-36) and *discoid plaques* as in CCLE (see Fig. 14-37), predominantly on the face and on the arms and

Laboratory Examinations

Pathology. Skin. Atrophy of epidermis, liquefaction degeneration of the dermal–epidermal junction, edema of the dermis, dermal lymphocytic infiltrate, and fibrinoid degeneration of the connective tissue and walls of the blood vessels.

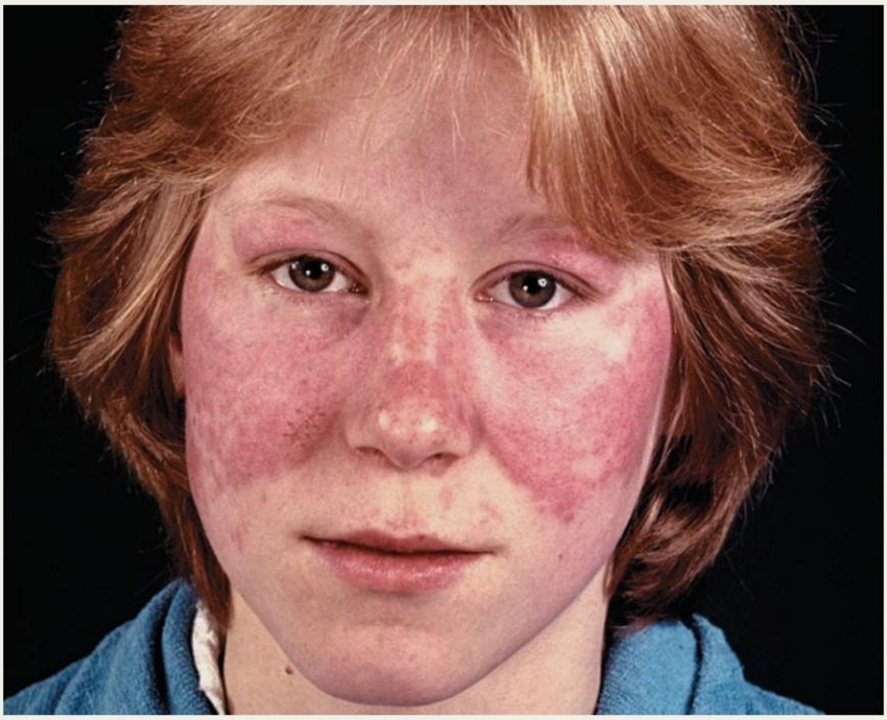


Figure 14-33. Acute systemic lupus erythematosus Bright red, sharply defined erythema with slight edema and minimal scaling in a "butterfly pattern" on the face. This is the typical "malar rash." Note also that the patient is female and young.

Immunofluorescence of Skin. The lupus band test (LBT, direct immunofluorescence) shows granular or globular deposits of IgG, IgM, C3 in a band-like pattern along the dermal–epidermal junction. Positive in lesional skin in 90% and in the clinically normal skin (sun exposed, 70–80%; non–sun exposed, 50%).

Serology. ANA positive (>95%); peripheral pattern of nuclear fluorescence. Anti–double-strand DNA antibodies, anti-Sm antibodies, and rRNP antibodies specific for SLE; low levels of complement (especially with renal involvement). Anticardiolipin autoantibodies (lupus anticoagulant) in a specific subset (anticardiolipin syndrome); SS-A(Ro) autoantibodies have a low specificity for SLE but are specific in the subset of SCLE (see below) (Table 14-4).

Hematology. Anemia [normocytic, normochromic, or, rarely, hemolytic Coombs-positive,

TABLE 14-4 PATHOGENIC AUTOANTIBODIES IN SYSTEMIC LUPUS ERYTHEMATOSUS

Skin	Anti-double-strand DNA (70–80%) Nucleosome (60–90%) Ro (30–40%)
Brain	NMDA receptor (33–50%)
Kidney	Anti-double-strand DNA (70–80%) Nucleosome (60–90%) C1q (40–50%) Ro (30–40%) Sm (10–30%) Alpha-actinin (20–30%)
Thrombosis	Phospholipids (20–30%)
Fetal cardiac abnormalities	Ro (30–40%) La (15–20%)
Pregnancy loss	Phospholipids (20–30%)

14-4).

Hematology. Anemia [normocytic, normochromic, or, rarely, hemolytic Coombs-positive,

abnormalities	La (15–20%)
Pregnancy loss	Phospholipids (20–30%)

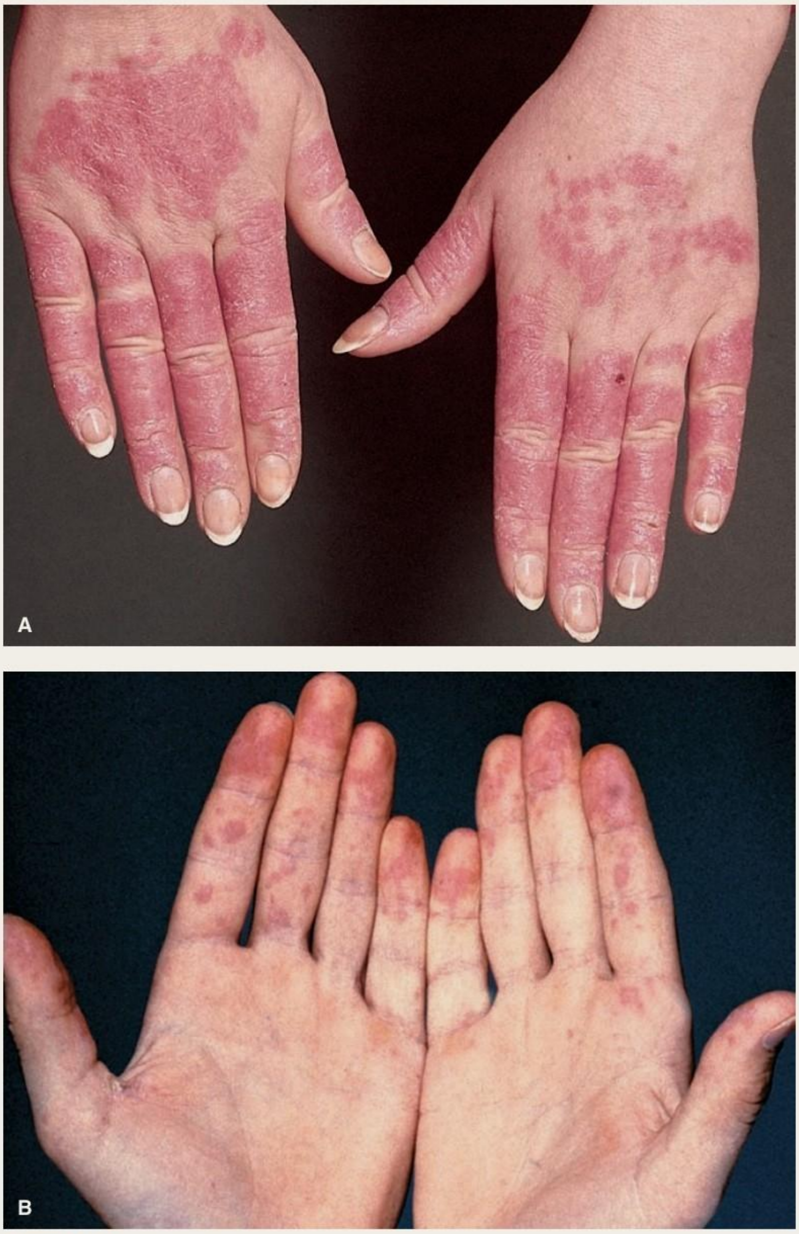


Figure 14-34. Acute SLE (A) Red-to-violaceous, well-demarcated papules and plaques on the dorsa of the fingers and hands, characteristically sparing the skin overlying the joints. This is an important differential diagnostic sign when considering dermatomyositis, which characteristically involves the skin over the joints (compare with Fig. 14-29B). **(B)** Palmar erythema mainly on the fingertips. This is pathognomonic.

leukopenia (>4000/μL)], lymphopenia, thrombocytopenia, elevated ESR.

Urinalysis. Persistent proteinuria, casts.

Management

General Measures. Rest, avoidance of sun exposure.

Indications for Prednisone (60 mg/d in divided

Periungual telangiectasia, diffuse nonscarring alopecia.

- *Distribution:* Scattered, disseminated in light-exposed areas—shoulders, extensor surface of

but not for systemic disease. Hydroxychloroquine 400 mg/d, quinacrine hydrochloride 100 mg/d. In systemic involvement prednisone ± immunosuppressants.

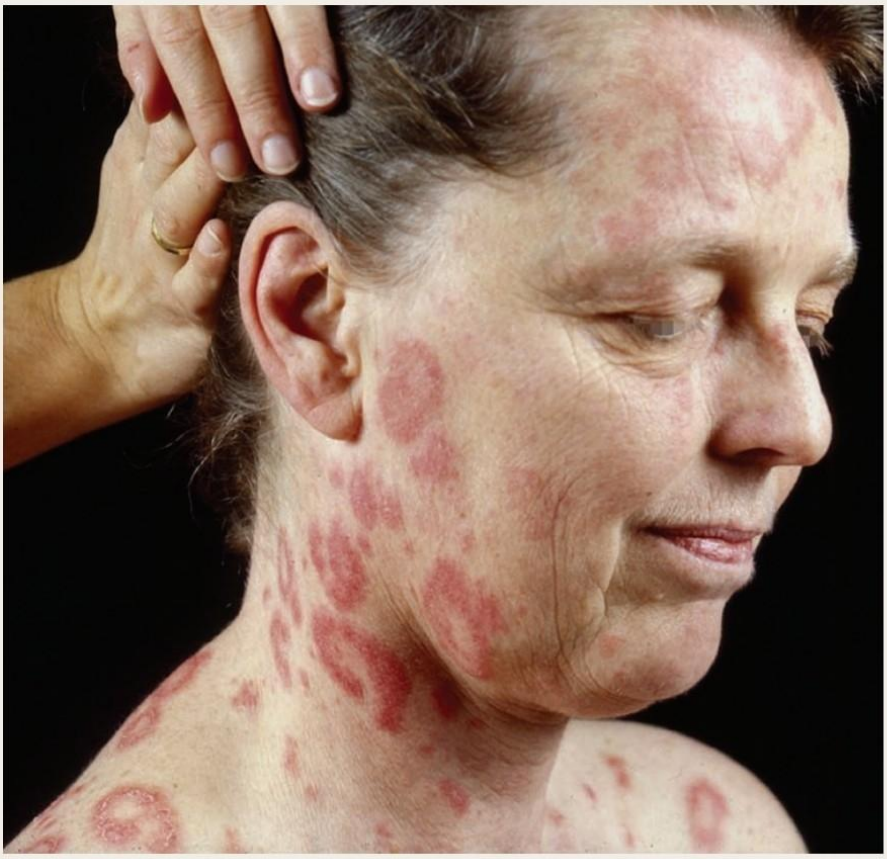


Figure 14-36. Subacute cutaneous lupus erythematosus Round, oval, and annular red plaques on the forehead, cheeks, neck, and upper trunk that show, but minimal, scaling in a 56-year-old woman. The eruption occurred after solar exposure. This is the annular type of SCLE.

Chronic Cutaneous Lupus Erythematosus (CCLE) ☐ ◐

ICD-9: 695.4 ∘ ICD-10: L93.0

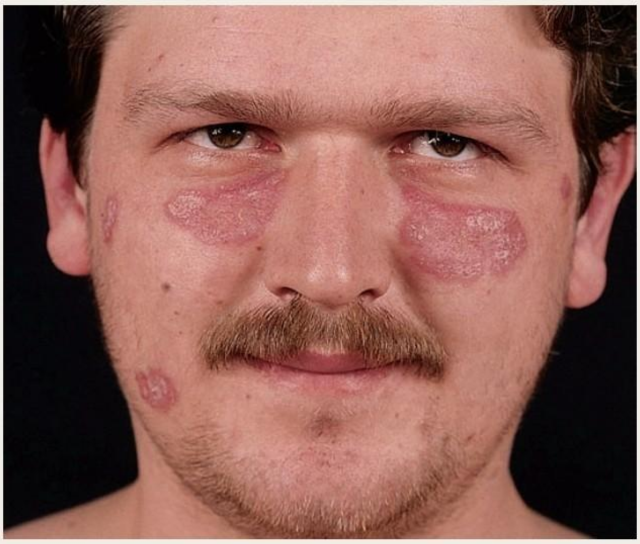


Figure 14-37. Chronic cutaneous lupus erythematosus Well-demarcated, erythematous, hyperkeratotic plaques with atrophy, follicular plugging, and adherent scale on both cheeks. This is the classic presentation of chronic discoid LE.

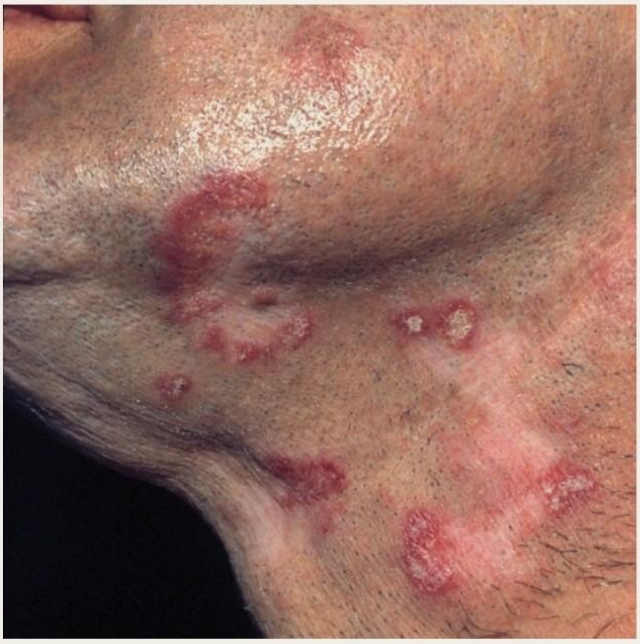


Figure 14-38. Chronic cutaneous lupus erythematosus: scarring There are multiple scarred lesions that are white and depressed and at their margins have active erythematous and scaly lesions. This can be quite disfiguring.

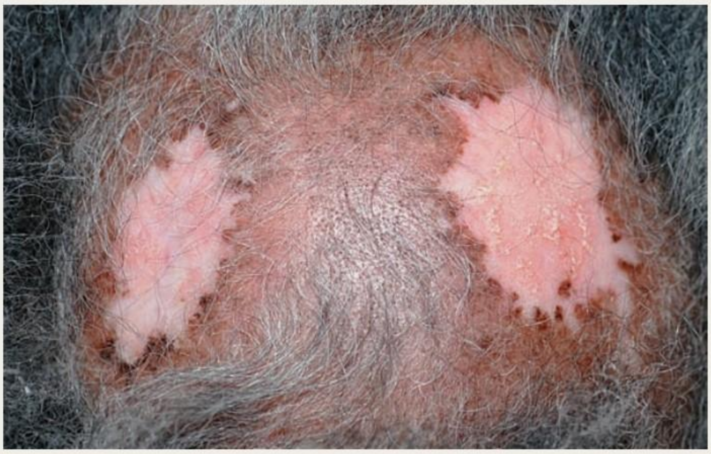


Figure 14-39. Chronic cutaneous lupus erythematosus Involvement of the scalp has led to complete hair loss with residual erythema, atrophy, and white scarring in this black male. Sharp demarcation of the lesions in the periphery indicates that these lesions originally were CDLE plaques.

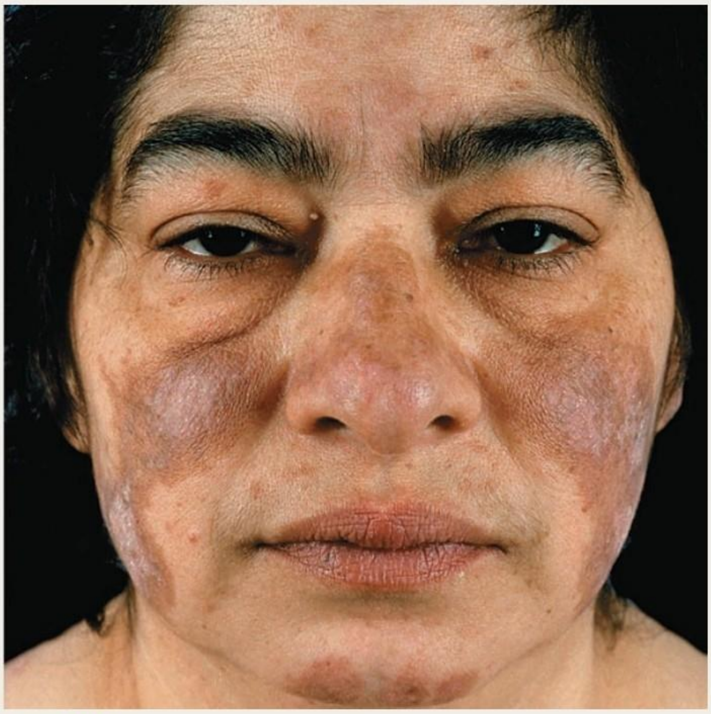


Figure 14-40. Chronic cutaneous lupus erythematosus: hyperpigmentation As inflammatory lesions resolve, there may be hyperpigmentation of the atrophic and partially scarred lesional skin, particularly in SPT III and IV patients. Although the skin lesions were CCLE, the patient had SLE.

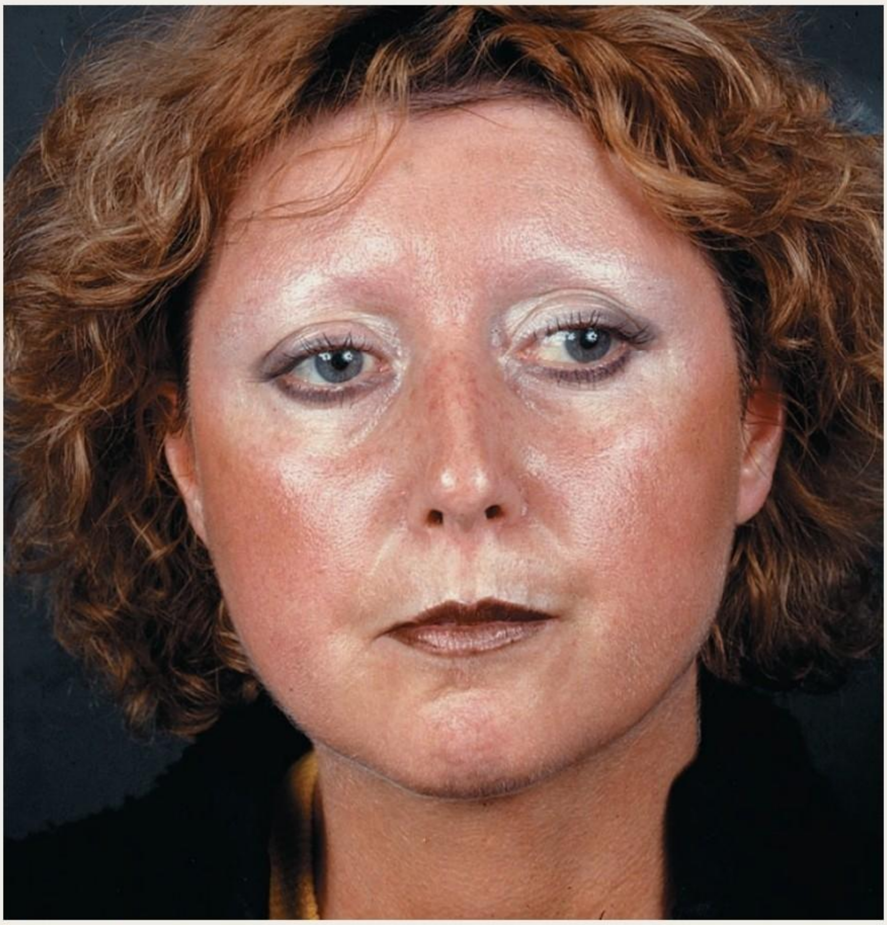


Figure 14-47. **Scleroderma (dSSc)** Mask-like facies with stretched, shiny skin and loss of normal facial lines giving a younger appearance than actual age; the hair is dyed. Thinning of the lips and perioral sclerosis result in a small mouth. Sclerosis (whitish, glistening areas) and multiple telangiectases (not visible at this magnification) are also present.

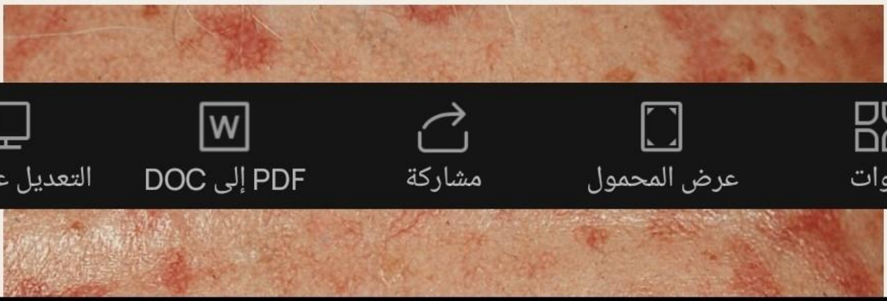
broadening and homogenization of collagen bundles, obliteration and decrease of interbundle spaces, thickening of dermis with replacement of upper or total subcutaneous fat by hyalinized collagen. Paucity of blood vessels, thickening/hyalinization of vessel walls.

Autoantibodies. Patients with dSSc have circulating ANA. Autoantibodies react with centromere proteins or DNA topoisomerase I; fewer patients have antinuclear antibodies. Anticentromeric autoantibodies occur in 21% of dSSc and 71% of CREST patients, DNA topoisomerase I (Scl-70) antibodies in 33% of dSSc and 18% of CREST patients.

Diagnosis and Differential Diagnosis

Clinical findings confirmed by dermatopathology.

Differential Diagnosis. *Diffuse sclerosis*: mixed connective tissue disease, eosinophilic fasciitis, scleromyxedema, morphea, porphyria cutanea tarda, chronic GVHD, lichen sclerosus et atrophicus, polyvinyl chloride exposure, adverse



Linear: Usually on extremity (Fig. 14-51) or *frontoparietal*—scalp and face (Fig. 14-52); here, it may resemble a scar from a strike with a saber (*en coup de sabre*).
Macular: Small (<3 mm) macular patches, confluent (Fig. 14-53A); clinically indistinguishable from lichen sclerosus et atrophicus (see p. 355).
Atrophic: Atrophoderma of Pasini and Pierini (Fig. 14-53B).
Pansclerotic: On trunk (Fig. 14-54) or extremities.

Mouth. With linear morphea of head, may have associated hemiatrophy of tongue.

Hair and Nails. Scarring alopecia with scalp plaque. Particularly with linear morphea of the head. Nail dystrophy in linear lesions of extremity or in pansclerotic morphea.

General Examination

Morphea around joints and linear morphea may lead to flexion contractures. Pansclerotic morphea is associated with atrophy and fibrosis of muscle. Extensive involvement of trunk may result in restricted respiration. With linear morphea of the head (Fig. 14-52), there may be associated atrophy of ocular structures and atrophy of bone. *Note:* morphea may be associated with lichen sclerosus et atrophicus.

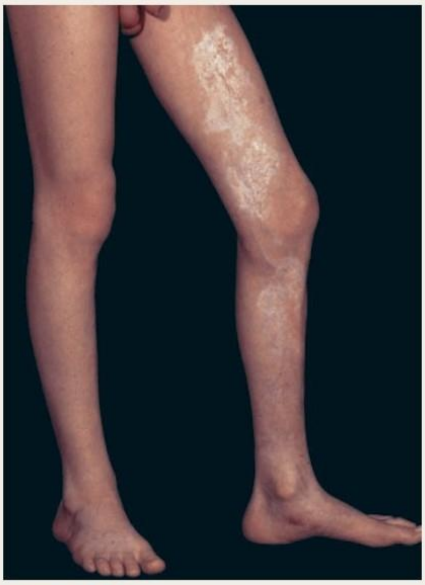
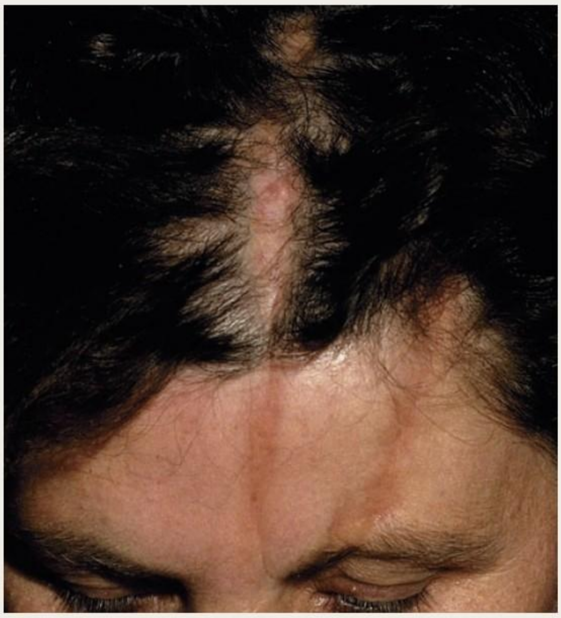


Figure 14-51. Linear Morphea Indurated, ivory-white lesion extending from upper thigh to the dorsum of the foot. Induration is pronounced, and in the region above the knee it extends to the fascia (pansclerotic morphea). If progressive, it will limit the movement of the joint.

Figure 14-52. Linear morphea, "en coup de sabre" Two linear, partially ivory-white (on the scalp) and hyperpigmented (on the forehead) depressed lesions extending from the crown of the head, where they have led to alopecia, over the forehead to the orbita. They look like scars after strikes with a saber, hence the French designation. These lesions can extend to the bone and rarely to the dura mater.



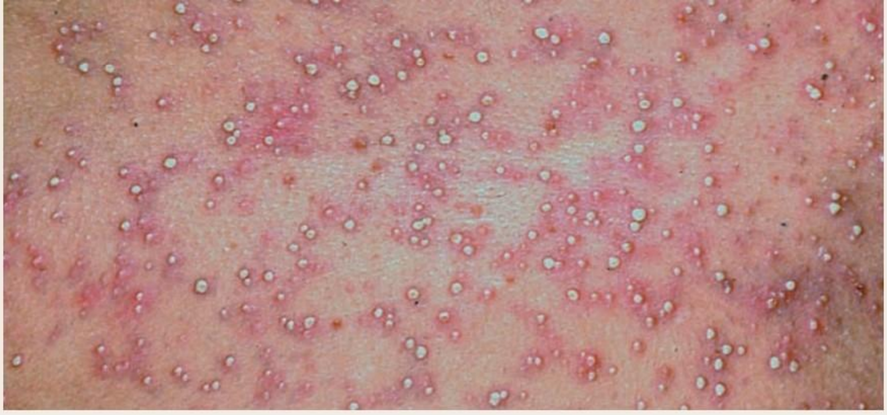


Figure 23-2. **Pustular drug eruption: acute generalized exanthematous pustulosis (AGEP)** Multiple tiny nonfollicular pustules against the background of diffuse erythema that first appeared in the large folds and then covered the entire trunk and the face.

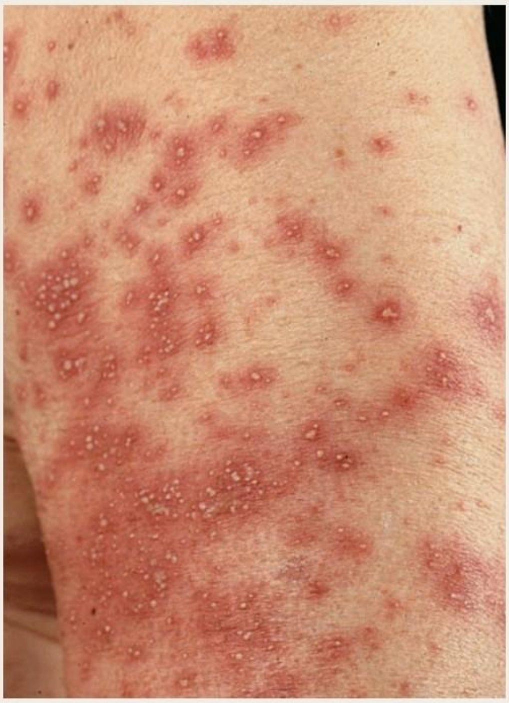


Figure 23-3. **Pustular drug eruption: AGEP** Multiple sterile pustules surrounded by fiery-red erythema in a 58-year-old female who had fever and leukocytosis. In contrast to the disseminated pustules in Fig. 23-2, here the pustules show a tendency for grouping and confluence. Differential diagnosis of von Zumbusch pustular psoriasis (compare with Fig. 3-13).

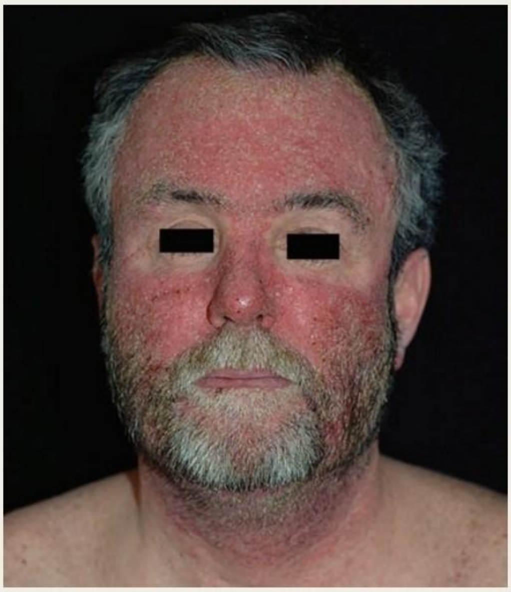


Figure 23-4. **Pustular drug eruption: erlotinib** This pustular eruption occurred in a patient who had received an anti-EGR monoclonal antibody for cancer of the colon localized to face. Differential diagnosis to acne and rosacea.

- Usually quite pruritic. Painful skin lesions suggest development of a more serious ACDR, such as toxic epidermal necrolysis (TEN).
- **Systems Review.** ± Fever, chills.
- **Skin Lesions.** Macules and/or papules, a few millimeters to 1 cm in size (Fig. 23-1). Bright or "drug" red. Resolving lesions have hues of tan

or IV Provides symptomatic relief. If offending drug cannot be substituted or omitted, systemic glucocorticoids can be administered to treat the ACDR. **Prevention.** Patients must be aware of their specific drug hypersensitivity and that other drugs of the same class can cross-react. Wearing a medical alert bracelet is advised.

Reactions to Specific Drugs (Selected)

Allopurinol. Incidence: 5%. Begins on face, spreads rapidly to all areas; may occur in photodistribution. Onset: 2–3 weeks after initiation of therapy. Associated findings: facial edema; systemic vasculitis, especially involving kidneys. Rash may fade in spite of continued administration.
Ampicillin, Amoxicillin. In up to 100% of patients with EBV or CMV mononucleosis syndrome. Increased incidence of EDR to penicillins in patients taking allopurinol. Ten percent cross-react with cephalosporins.

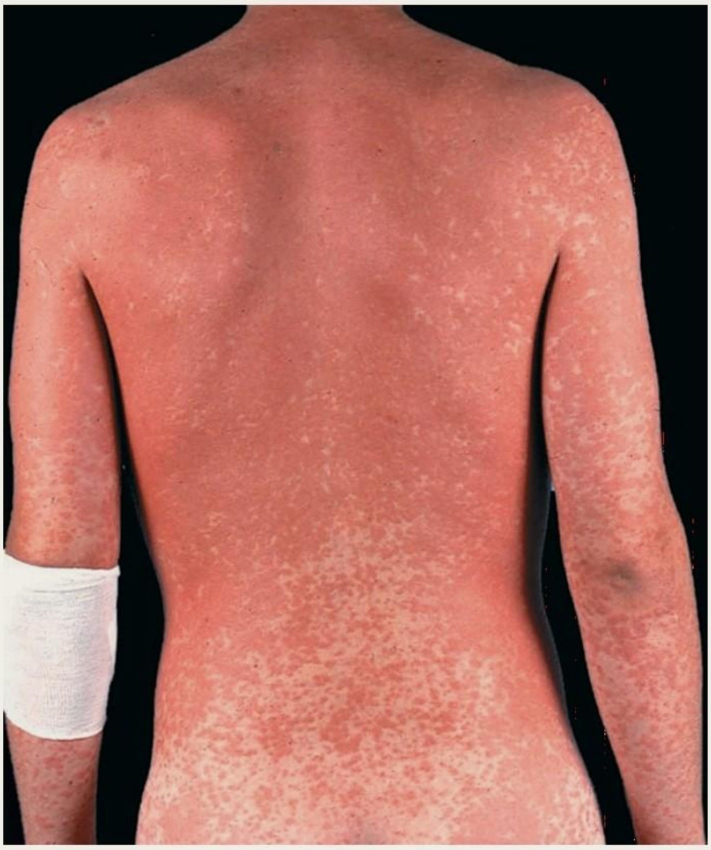


Figure 23-1. Exanthematous drug eruption: ampicillin Symmetrically arranged, brightly erythematous macules and papules, discrete in some areas, and confluent in others, on the trunk and the extremities.

Barbiturates. Site: face, trunk. Onset: few days after initiation of therapy. Cross-reactivity with other barbiturates: not universal.
Benzodiazepines. Rare. Onset: few days after initiation of therapy. Rechallenge: frequently rash does not occur.
Carbamazepine. Morphology: diffuse erythema; severe erythroderma may follow. Site: begins on face, spreads rapidly to all areas; may occur in photodistribution. Onset: 2 weeks after initiation of therapy. Associated findings: facial edema.
Gold Salts. Incidence: 10–20% of patients; dose-related. Morphology: diffuse erythema; exfoliative dermatitis, lichenoid, hemorrhagic, bullous, or pityriasis rosea-like eruptions may follow.
Hydantoin Derivatives. Macular → confluent erythema. Begins on face, spreads to trunk and extremities. Onset: 2 weeks after initiation of therapy. Associated findings: fever, peripheral eosinophilia; facial edema; lymphadenopathy (can mimic lymphoma histologically).
Isoniazid. May evolve to exfoliative dermatitis. Associated findings: fever and hepatitis.
Phenothiazines. Begins on face, spreads to trunk (mainly back), and extremities. Onset: between second and third weeks after initiation of therapy. Associated findings: periorbital edema. Rechallenge: rash may not occur. Cross-reactivity: common.
Sulfonamides. Occurs in up to 50–60% of HIV/AIDS-infected patients (trimethoprim sulfamethoxazole). Patients sensitized to one sulfa-based drug may cross-react with another sulfa drug in 20%.

pruritic, painful, or burning. *Time to onset of lesion(s)*: Occur from 30 min to 8 h after ingestion of drug in previously sensitized individual. *Duration of lesion(s)*: Lesions persist if drug is continued. Resolve days to few weeks after drug is discontinued.

- **Skin Lesions.** A sharply demarcated macule, round or oval in shape, occurring within hours after ingestion of the offending drug. Initially erythema, then dusky red to violaceous (Fig. 23-6A). Most commonly, lesions are solitary and can spread to become quite large, but they may be multiple (Fig. 23-7) with random distribution. Lesions may
- **Patch Test.** Suspected drug can be placed as a patch test at a previously involved site; an inflammatory response occurs in only 30% of cases.
- FDE resolves within a few weeks of withdrawing the drug. Recurs within hours after ingestion of a single dose of the drug.
- **Management.** Withhold offending drug. Noneroded lesions: potent topical glucocorticoid ointment. Eroded lesions: antimicrobial ointment. For widespread, generalized, and highly painful mucosal lesions, oral prednisone 1 mg/kg body weight tapered over a course of 2 weeks.

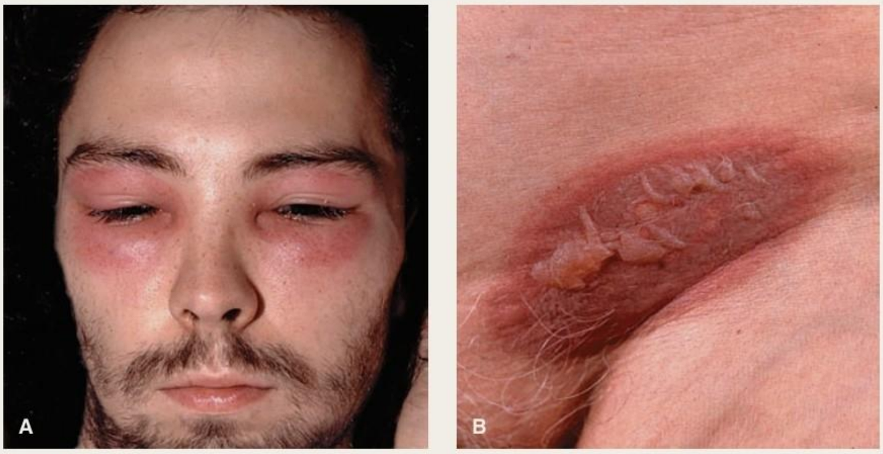


Figure 23-6. **Fixed drug eruption (A)** Tetracycline. Two well-defined periorbital plaques with edema. This was the second such episode following ingestion of a tetracycline. No other lesions were present. **(B)** Tylenol. A large oval violaceous lesion with blistering in the center. Erosive mouth lesions were also present.

TABLE 23-6 MOST COMMONLY IMPLICATED AGENTS IN FIXED DRUG ERUPTIONS

Tetracyclines (tetracycline, minocycline)
Sulfonamides, other sulfa drugs
Metronidazole, nystatin, salicylates, NSAIDs, phenylbutazone, phenacetin
Barbiturates
Oral contraceptives
Quinine (including quinine in tonic water), quinidine
Phenolphthalein
Food coloring (yellow): in food or medications

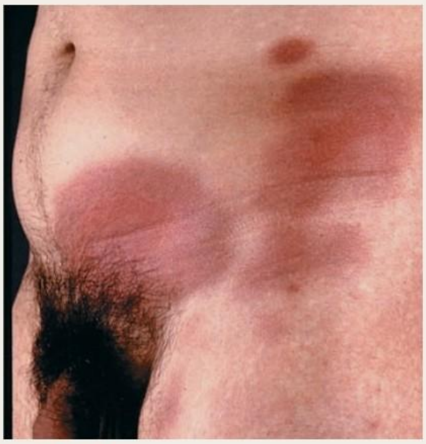


Figure 23-7. **Fixed drug eruption** Doxycycline. Multiple lesions. Similar violaceous plaques were also on the anterior and posterior trunk.

Drug Hypersensitivity Syndrome ◧ ◐ → ○

ICD-9: 995.2 ◦ ICD-10: I88.7

- Drug-induced alterations in pigmentation are relatively common.
- They result from the deposition of a variety of endogenous and exogenous pigments in the skin.
- Can be of significant cosmetic concern to the patient.
- Drugs most commonly causing hyperpigmentation:
  - Antiarrhythmetic: amiodarone
  - Antimalarial: chloroquine, hydroxychloroquine, quinacrine, quinine
  - Antimicrobial: minocycline, clofazimine, zidovudine
  - Antiseizure: hydantoins
  - Cytostatic: bleomycin, cyclophosphamide, doxorubicin, daunorubicin, busulfan, 5-fluorouracil, dactinomycin
  - Metals: silver, gold, iron
  - Hormones: ACTH estrogen/progesterone
  - Psychiatric: chlorpromazine
  - Dietary: β-carotene

Clinical Manifestation

Amiodarone. More than 75% of patients after 40-g cumulative dose after >4 months of therapy. More common in skin phototypes I and II. Low-grade or minimal photosensitivity; phototoxic erythema limited to the light-exposed areas in a small proportion (8%) of patients. Dusky-red erythema and, later, blue-gray dermal melanosis (Fig. 23-9) in exposed areas (face and hands). Lipofuscin-type pigment deposited in macrophages and endothelial cells.

Antimalarials. *Cloroquine, hydroxychloroquine*. Occurs in 25% of individuals who take the drug for >4 months. Brownish, gray-brown, and/or blue-black discoloration due to melanin, hemosiderin. Over shins; face, nape of neck; hard palate (sharp line of demarcation at soft palate); under finger- and toenails (see Section 34); may also occur in cornea and retina; *Quinacrine*: yellow, yellow-green skin, and sclerae (resembling icterus); yellow-green fluorescence of nail bed with Wood lamp.

Minocycline. Onset delayed, usually after total dose of >50 g, but may occur after a small dose. Not melanin but an iron-containing brown pigment, located in the dermal macrophages; stippled or diffuse. Blue-gray or slate-gray pigmentation (Fig. 23-10). Distributed on extensor legs, ankles, dorsa of feet, face, especially around eyes; sites of trauma or inflammation such as acne scars, contusions, abrasions; hard palate, teeth; nails. **Clofazimine.** Orange, reddish brown (range, pink to black) discoloration, ill-defined on light-exposed areas; conjunctivae;

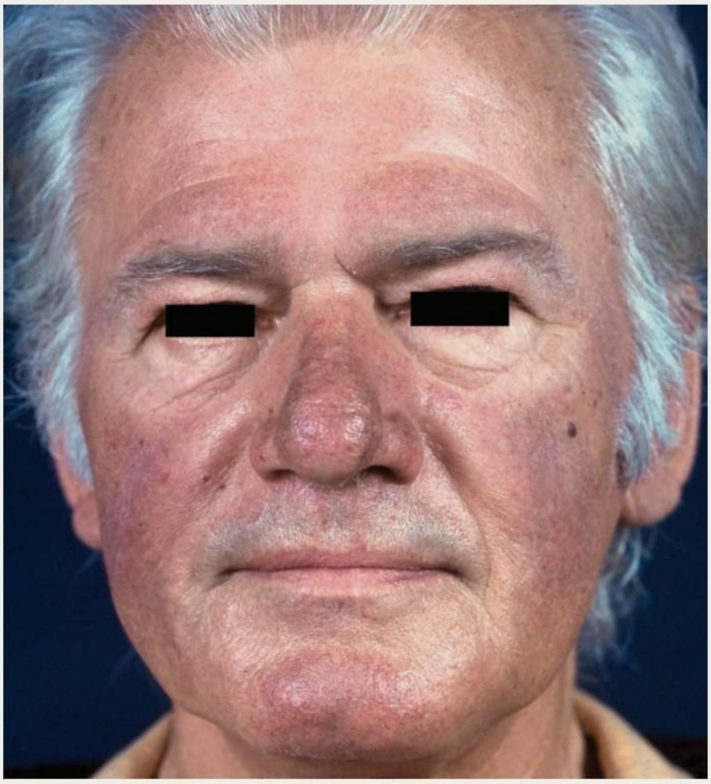

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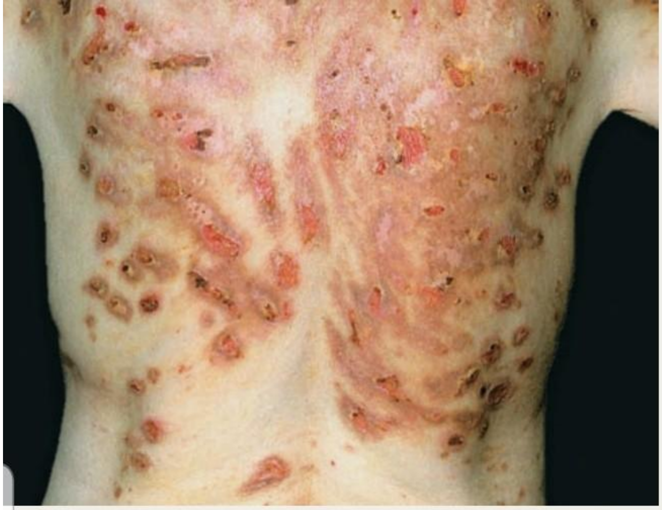
Figure 23-9. Drug-induced pigmentation: amiodarone A striking mix of a slate gray and brown pigmentation in the face. The bluish color is due to the deposition of melanin and lipofuscin contained in macrophages and endothelial cells in the dermis. The brown color is due to melanin. The pigmentation is reversible, but it may take up to a year or more to complete resolution. In this patient, it took 16 months for the pigmentation to disappear.



10 years. The ulcerated crusted lesion resolved with cloth tape occlusion. Once the protection was removed, the patient resumed excoriating the sites.

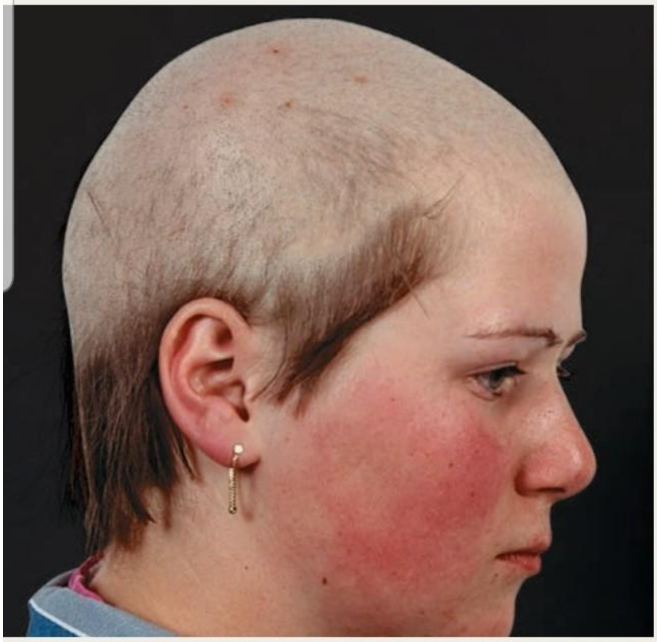


Figure 24-4. Trichotillomania This extensive alopecia has resulted from pulling and plucking hairs by the 17-year-old patient. She appeared balanced but mildly depressed and had considerable conflict with her parents. She admitted pulling hairs after considerable questioning.

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actitious Syndromes (Münchhausen Syndrome) ◧ ◐
CD-9: 301.51 ∘ ICD-10: F 68.1

- The term *factitious* means "artificial," and in this condition there is a self-induced dermatologic lesion(s); either the patient claims no responsibility or admits deliberately mutilating the skin.
- It occurs in young adults, females > males. The history of the evolution of the lesions is vague ("hollow" history).
- The lesions may be present for weeks to months to years (Fig. 24-5).
- Patient may be normal looking and act normally in every respect, although frequently there is a strange affect and bizarre personality.
- The skin lesions consist of cuts (Fig. 24-5), ulcers, and dense adherent necrotic eschar (Fig. 24-6). The shape of the lesions may be linear
- It is important to rule out every possible cause—chronic infections, granulomas, and vasculitis—perform a biopsy before assigning the diagnosis of *dermatosis artefacta*, both for the benefit of the patient and because the physician may be at risk for malpractice if he or she fails to diagnose a true pathologic process.
- There is often serious personality and/or psychosocial stress, or a psychiatric disease.
- The condition demands the utmost tact on the part of the physician, who can avert a serious outcome (i.e., suicide) by attempting to gain enough empathy with the patient to ascertain the cause. This varies with the nature of the psychiatric problem.
- The condition may persist for years in a patient

Pyoderma is an archaic term, literally "pus in the skin." Skin and soft-tissue infections, commonly caused by *Staphylococcus aureus* and group A streptococcus (GAS), have been

Cutaneous production of toxins by bacteria (*S. aureus and* GAS) causes systemic intoxications such as toxic shock syndrome (TSS) and scarlet fever.

Erythrasma ICD-9: 039.0 ◦ ICD-10: L08.1 ☐ ●

Etiology. *Corynebacterium minutissimum*, gram-positive (diphtheroid) bacillus; normally in human microbiome. Growth favored by humid cutaneous microclimate.

Clinical Manifestation

Asymptomatic except for subtle discoloration.

Patches, sharply marginated (Fig. 25-1). Tan or pinkish; postinflammatory hyperpigmentation in more heavily pigmented individuals.

In webspaces of feet, may be macerated (Fig. 25-2). *Distribution:* intertriginous skin, i.e., toe webs (Fig. 25-2), inguinal folds, axillae, other occluded sites.

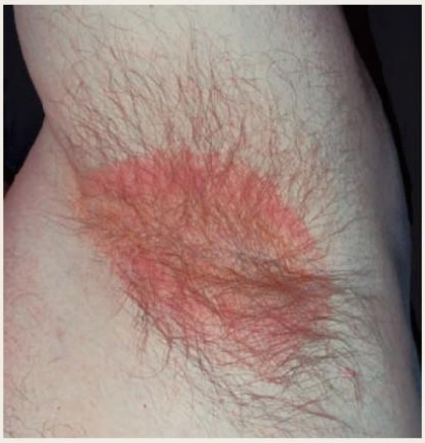


Figure 25-1. Erythrasma: axilla Sharply marginated, red patch in the axilla. Wood's lamp demonstrates bright coral-red, differentiating erythrasma from intertriginous psoriasis. KOH preparation was negative for hyphae.

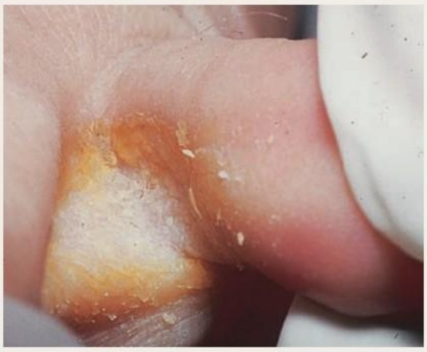


Figure 25-2. Erythrasma: webspace This macerated interdigital webspace appeared bright coral-red when examined with Wood's lamp; KOH preparation was negative for hyphae. The webspace is the most common site for erythrasma in temperate climates. In some cases, interdigital tinea pedis and/or pseudomonal intertrigo may coexist.

Diagnosis

Wood's lamp examination demonstrates coral-red fluorescence. KOH negative; rule out epidermal dermatophytosis.

Differential Diagnosis

Intertriginous psoriasis, epidermal dermatophytosis, pityriasis versicolor, Hailey–Hailey disease.

Course

Persists and recurs unless microclimate is altered.

Treatment

Usually controlled with benzoyl peroxide wash or sanitizing alcohol gel. Clindamycin lotion and erythromycin are beneficial.

Pitted Keratolysis ◧ ●

Etiology. *Kytococcus Sedentarius.* One of human microbiome on plantar feet in the setting of hyperhidrosis; produces two extracellular proteases that can digest keratin.

Clinical Manifestation

Punched out pits in stratum corneum, 1–8 mm in diameter (Fig. 25-3). Pits can remain discrete or become confluent, forming large areas of eroded stratum corneum. Lesions are more apparent with hyperhidrosis and maceration. Symmetric or asymmetric involvement of both feet. *Distribution:* Pressure-bearing areas, ventral aspect of toe, ball of foot, heel; interface of toes.

Diagnosis

Differential Diagnosis

Concomitant tinea pedis, erythrasma, candidal intertrigo, and pseudomonal webspace infection may be present.

Course

Persists and recurs unless microclimate is altered.

Treatment

Usually controlled with benzoyl peroxide

occi. Group A (Fig.
(Fig. 25-6). Strepto-
ogress to soft-tissue

- *S. aureus*. Often gains entry into skin via hair follicle, causing folliculitis and furuncles.
- *Pseudomonas aeruginosa* (Fig. 25-7).
- *C. minutissimum* (erythrasma) (Figs. 25-1 and 25-2). *K. sedentarius* (pitted keratolysis) (Fig. 25-3).

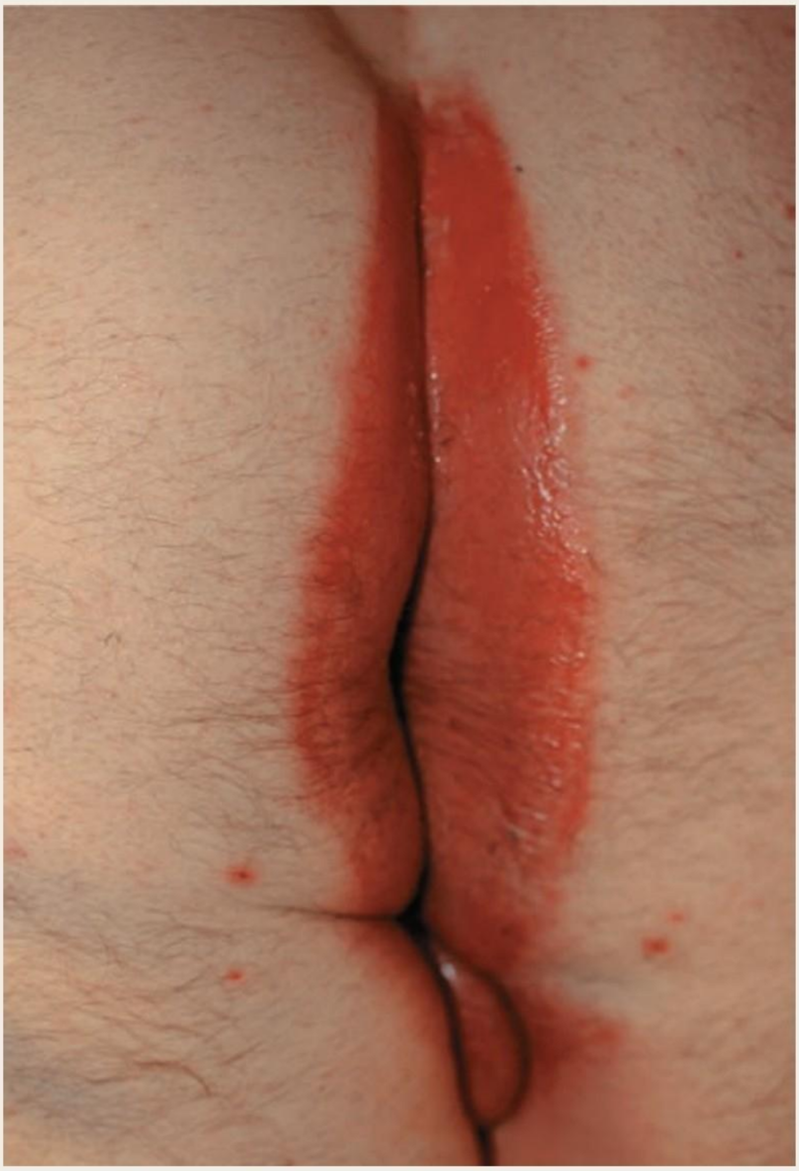


Figure 25-5. Intergluteal intertrigo: group A streptococcus A painful moist erythematous plaque in a male with intertriginous psoriasis, with foul odor. Infection resolved with penicillin VK.

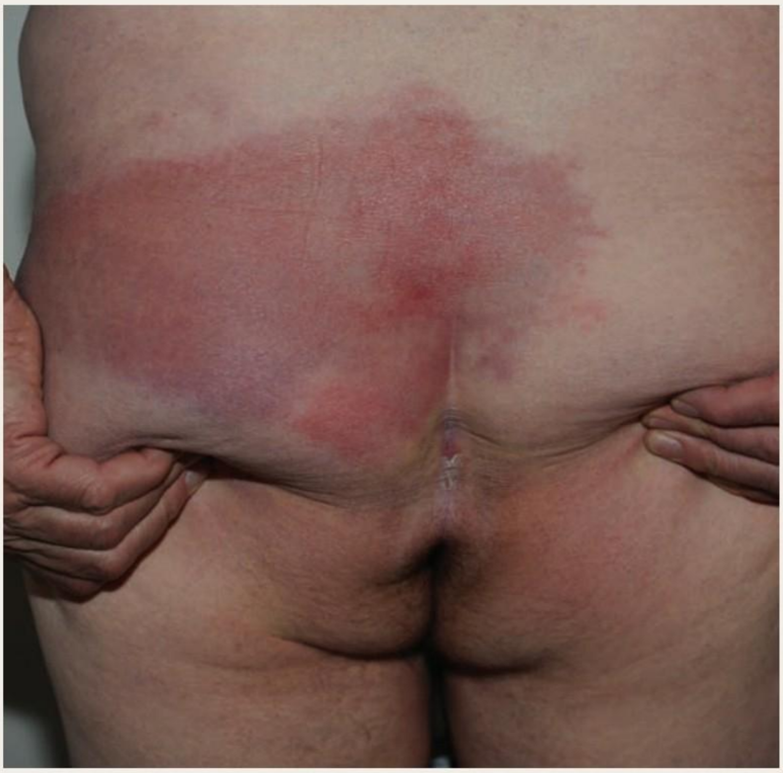


Figure 25-6. Erysipelas: group G streptococcus 65-year-old male with sharply marginated erythematous plaque on buttocks. Portal of entry of infection was intergluteal intertrigo.

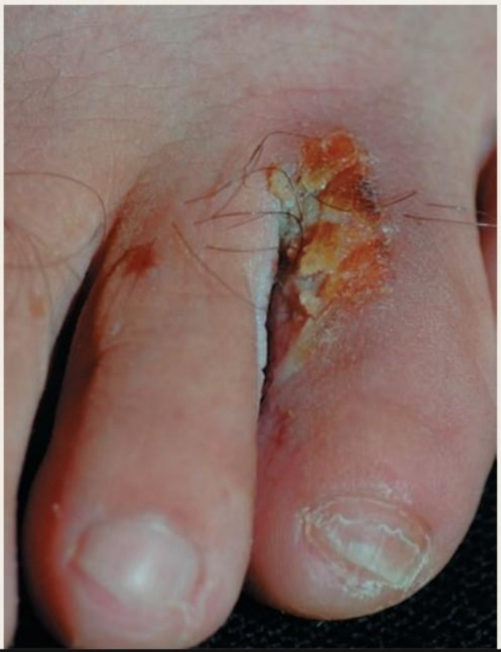


Figure 25-7. Webspace intertrigo: *P. aeruginosa* Erosion of a webspace of the foot with a bright red base and surrounding erythema. Tinea pedis (interdigital and moccasin patterns) and hyperhidrosis were also present, which facilitated growth of *Pseudomonas.*

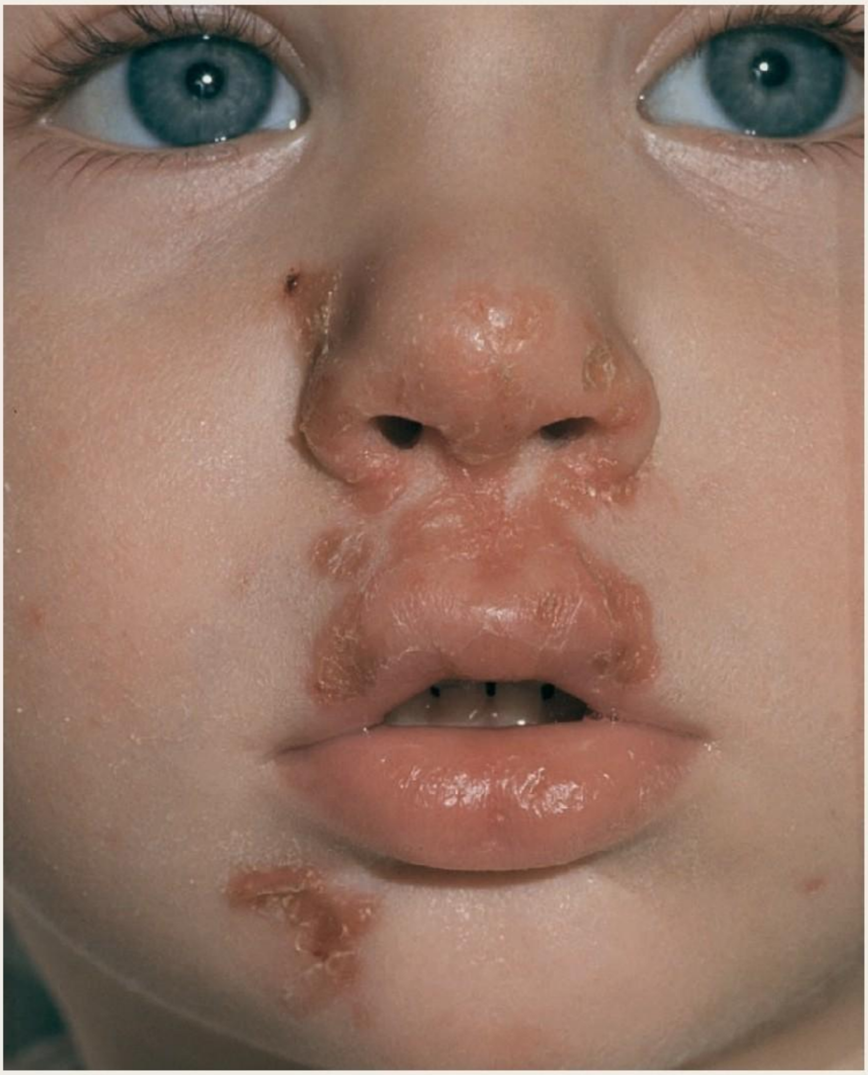


Figure 25-8. Impetigo: MSSA Crusted erythematous erosions becoming confluent on the nose, cheek, lips, and chin in a child with nasal carriage of *S. aureus* and mild facial eczema.

Differential Diagnosis

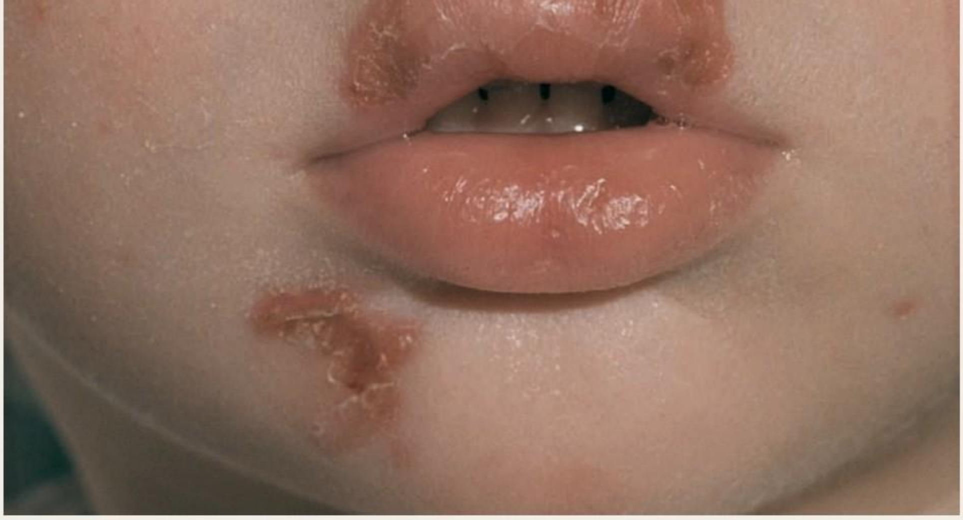
Impetigo. Excoriation, allergic contact dermatitis, herpes simplex, epidermal dermatophytosis, scabies. *Most erosions with "honey-colored crusts" are not impetigo.*

Intact Bullae. Acute allergic contact dermatitis, insect bites, thermal burns, porphyria cutanea tarda (PCT) (dorsa of hands).

Ecthyma. Excoriations, excoriated insect bites, PCT, venous (stasis) and ischemic ulcers (legs).

Diagnosis

Clinical findings confirmed by culture: *S. aureus*, commonly; failure of oral antibiotic suggests MRSA. GAS.



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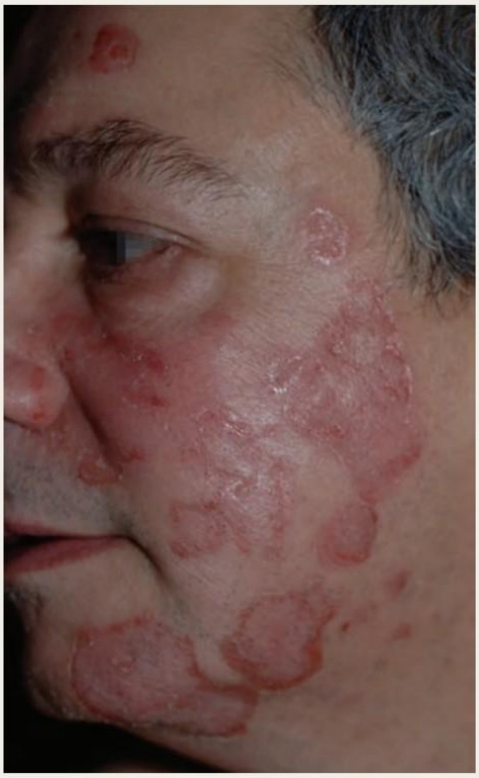


Figure 25-9. Impetigo: MRSA 45-year-old male with large crusted erosions, becoming confluent, with central clearing on the face. MRSA colonized the nares.

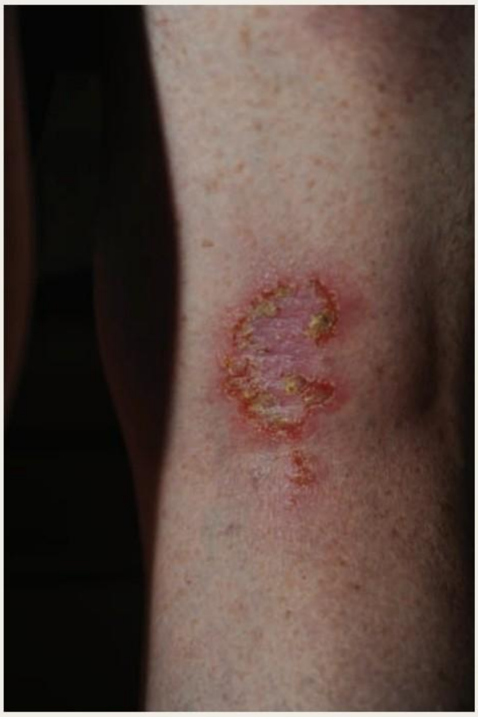


Figure 25-10. Secondary infection of Hailey–Hailey disease: MRSA 51-year-old female with Hailey–Hailey disease has chronic MRSA infection of cutaneous erosions on thigh.

Figure 25-11. Secondary infection of pemphigus foliaceus: MRSA 65-year-old female with recalcitrant pemphigus foliaceus has extensive infection of cutaneous erosions on the face.

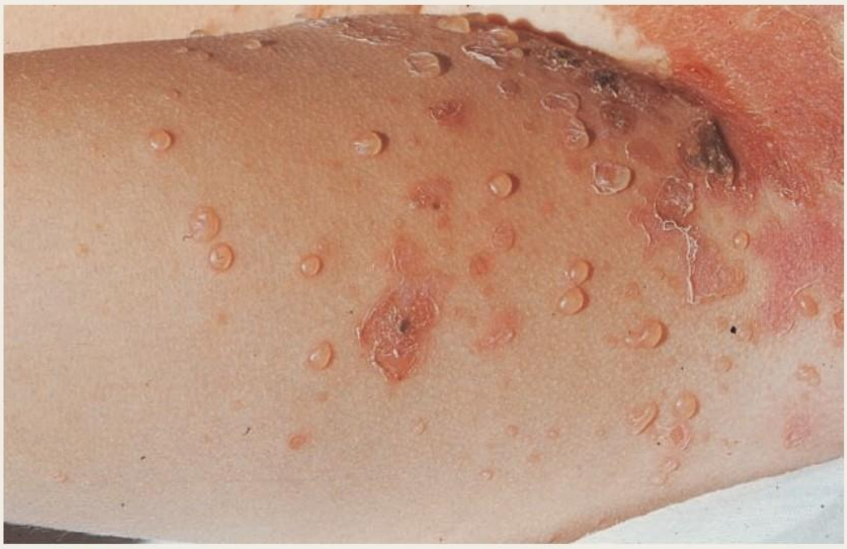


Figure 25-12. Bullous impetigo Scattered, discrete, intact, and ruptured thin-walled blisters on the inguinal area and adjacent thigh of a child; lesions in the groin have ruptured, resulting in superficial erosions.

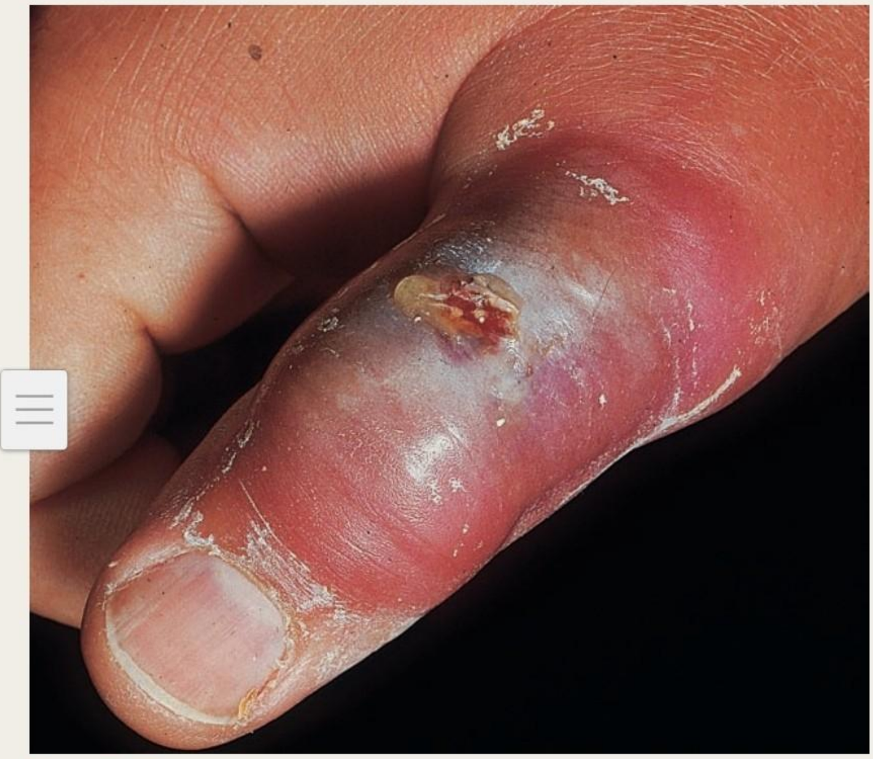
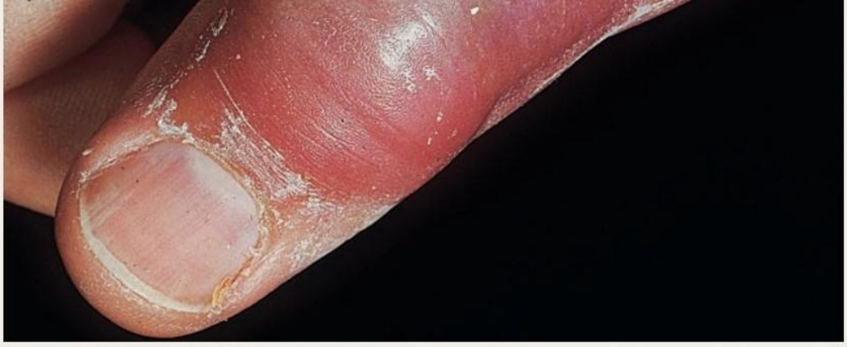


Figure 25-13. Bullous impetigo with blistering dactylitis: ***S. aureus*** A large, single bulla with surrounding erythema and edema on the thumb of a child; the bulla has ruptured and clear serum exudes.

Course

Untreated, lesions of impetigo become more extensive and ecthyma. With adequate treatment, prompt resolution. Lesions can progress to deeper skin and soft-tissue infections.



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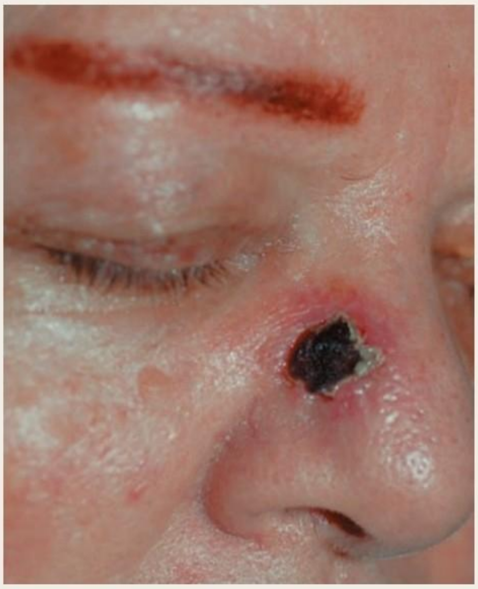

Figure 25-14. Ecthyma: MSSA Thickly crusted erosion/ulcer on the nose had been present for 6 weeks, arising at the site of a small wound. The crust was adherent and the site bled with debridement.

Course

Untreated, lesions of impetigo become mor extensive and ecthyma. With adequate treatment, prompt resolution. Lesions can progress to deeper skin and soft-tissue infections Nonsuppurative complications of GAS infection include guttate psoriasis, scarlet fever, an glomerulonephritis. Ecthyma may heal wit scarring. Recurrent *S. aureus* or GAS infection can occur because of failure to eradicate pathogen or by recolonization. Undiagnosed MRS infection does not respond to usual oral antibiotics given for methicillin-sensitive *S. aureus*

Treatment

Prevention. Benzoyl peroxide wash. Chec family members for signs of impetigo. Ethanol or isopropyl gel for hands and/or involve sites.

Topical Treatment. Mupirocin and retapamuli ointment is highly effective in eliminating *S aureus* from the nares and cutaneous lesions.

Systemic Antimicrobial Treatment. Accordin to sensitivity of isolated organism.

Abscess, Furuncle, Carbuncle
ICD-9: 680.9/682.9 ◦ ICD-10: L02

□ ◐

- Deeper skin infections can follow traumatic inoculation into skin or extension of infection into hair follicles.
- *Abscess*: Acute or chronic localized inflammation, associated with a collection of pus accumulated in a tissue. Inflammatory response to an infectious process or foreign material.
- *Folliculitis*: Infection of hair follicle with ± pus in the ostium of follicle (see Section 31).
- *Furuncle*: Acute, deep-seated, red, hot, tender nodule or abscess (boil) that evolves from a staphylococcal folliculitis.
- *Carbuncle*: Deeper infection composed of interconnecting abscesses usually arising in several contiguous hair follicles.

Epidemiology and Etiology

S. aureus (MSSA, MRSA).

Other Organisms. Much less common.

Sterile abscess can occur as a foreign-body response (splinter, ruptured inclusion cyst, injection sites). Cutaneous odontogenic sinus can appear anywhere on the lower face, even at sites distant from the origin (see Fig. 33-23).

Folliculitis, furuncles, and carbuncles repre-

Clinical Manifestation

Folliculitis may be slightly tender. With deepe infection, pain and tenderness. Carbuncles may be accompanied by low-grade fever and malaise; lesions are red, hot, and painful/tender.

Abscess. May arise in any organ or tissue Abscesses that present on the skin arise in th dermis, subcutaneous fat, muscle, or a variety o deeper structures. Initially, a tender red nodul response (splinter, ruptured inclusion cyst, injection sites). Cutaneous odontogenic sinus can appear anywhere on the lower face, even at sites distant from the origin (see Fig. 33-23).

Folliculitis, furuncles, and carbuncles represent a continuum of severity of *S. aureus* infection. Portal of entry: ostium of hair follicle.

aise; lesions are red, hot, and painful/tender.

Abscess. May arise in any organ or tissue. Abscesses that present on the skin arise in the dermis, subcutaneous fat, muscle, or a variety of deeper structures. Initially, a tender red nodule forms. In time (days to weeks), pus collects within a central space (Fig. 25-15). A well-formed abscess is characterized by fluctuance of the central portion of the lesion. Arise at sites of trauma. Ruptured inclusion cyst on the back often present as painful abscess. When arising from *S. aureus* folliculitis, may be solitary or multiple.

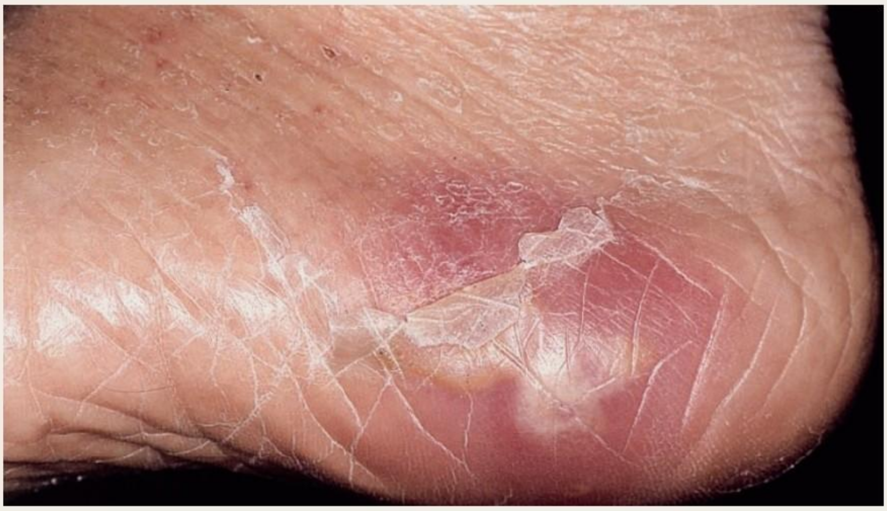

Figure 25-15. Abscess: MSSA A very tender abscess with surrounding erythema on the heel. The patient was a diabetic patient with sensory neuropathy; puncture by a sewing needle that was imbedded in the heel had provided a portal of entry. The foreign body was removed surgically.

Folliculitis (Staphylococcal). See "Infectious Folliculitis" in Section 31.

Furuncle. Initially, a firm tender nodule, up to 1–2 cm in diameter. In many individuals, furuncles occur in setting of staphylococcal folliculitis. Nodule becomes fluctuant, with abscess formation ± central pustule. Nodule with cavitation remains after drainage of abscess. A variable zone of cellulitis may surround the furuncle. *Distribution:* any hair-bearing region—beard area, posterior neck and occipital scalp, axillae, buttocks. Solitary or multiple lesions (Figs. 25-16 to 25-20).

Carbuncle. Evolution is similar to that of furuncle. Composed of several to multiple, adjacent, coalescing furuncles (Fig. 25-21). Characterized by multiple loculated dermal and subcutaneous abscesses, superficial pustules, necrotic plugs, and sieve-like openings draining pus.

Differential Diagnosis

Painful Dermal/Subcutaneous Nodule. Ruptured epidermoid or pilar cyst, hidradenitis suppurativa (axillae, groin, vulva).

Diagnosis

Clinical findings confirmed by findings on Gram staining and culture.

Course

Most abscesses resolve with effective treatment. If diagnosis and treatment are delayed, furunculosis can be complicated by soft-tissue infection, bacteremia, and hematogenous seeding of viscera. Some individuals are subject to recurrent furunculosis, particularly diabetics.

Treatment

The treatment of an abscess, furuncle, or carbuncle is incision and drainage plus systemic antimicrobial therapy.

axillae, buttocks. Solitary or multiple lesions (Figs. 25-16 to 25-20).

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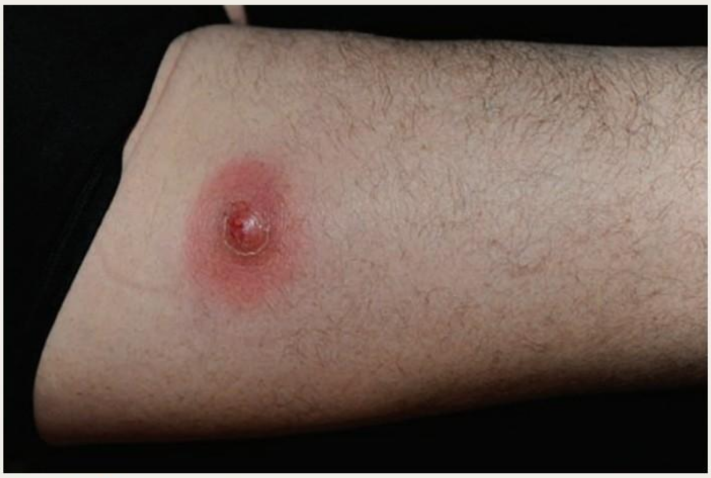

Figure 25-16. Furuncle: MSSA Abscess on the medial thigh of a 52-year-old male. The lesion was incised and drained and treated with doxycycline.

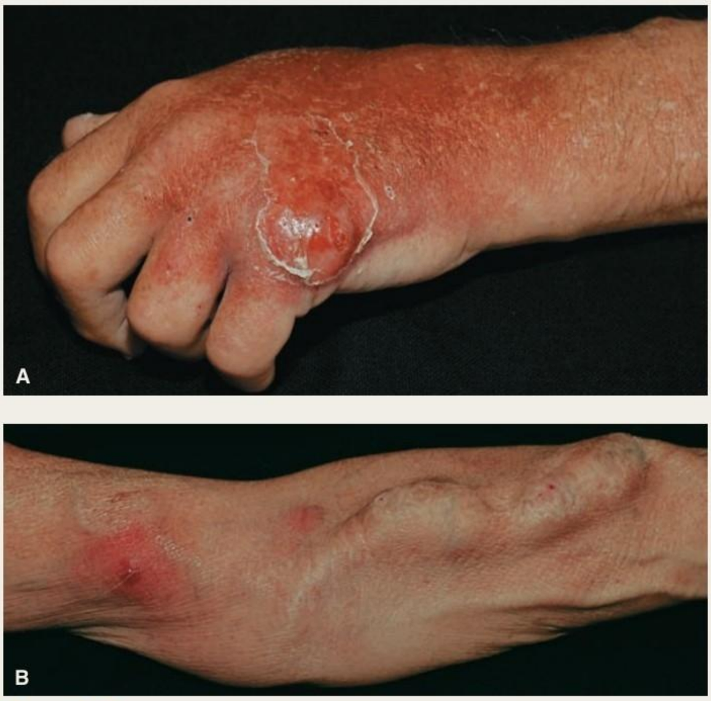

Figure 25-17. Furuncles and cellulitis: MRSA A 64-year-old male developed furuncles on the dorsum of the left hand **(A)** and forearm **(B)**. He had a fistula on his forearm and was dialyzed three times per week. Infection was spreading from the abscess with cellulitis.

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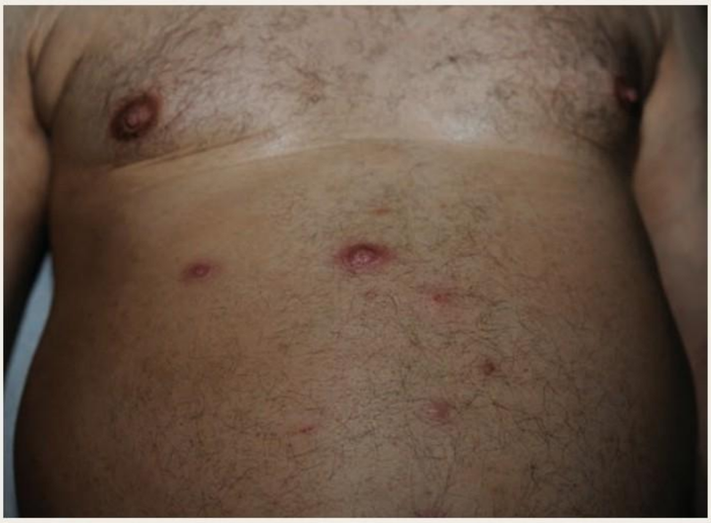


Figure 25-18. Multiple furuncles on the abdomen: MRSA 66-year-old operating room technician with multiple painful nodules. MRSA was isolated on culture of the nares and an abscess. He was treated with doxycycline, mupirocin to nares, and bleach baths. He was restricted from returning to work until cultured sites were negative for *S. aureus* colonization.

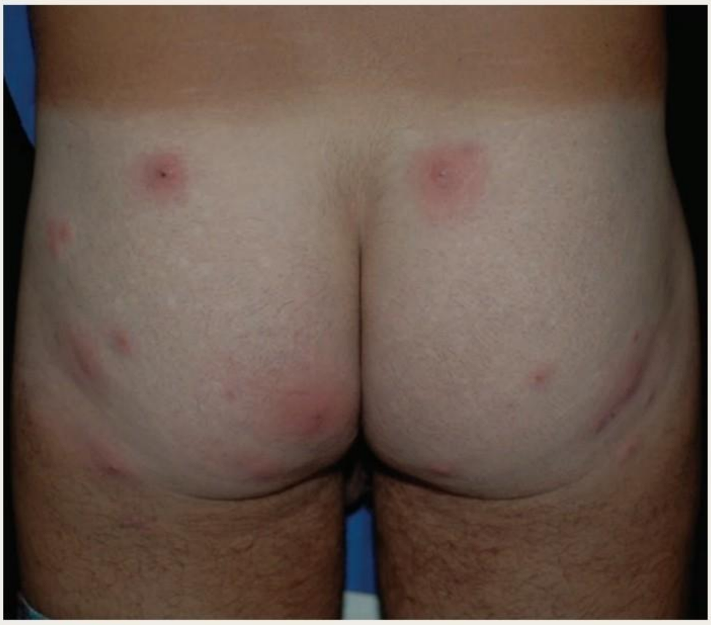
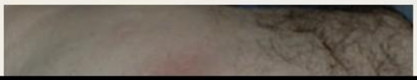
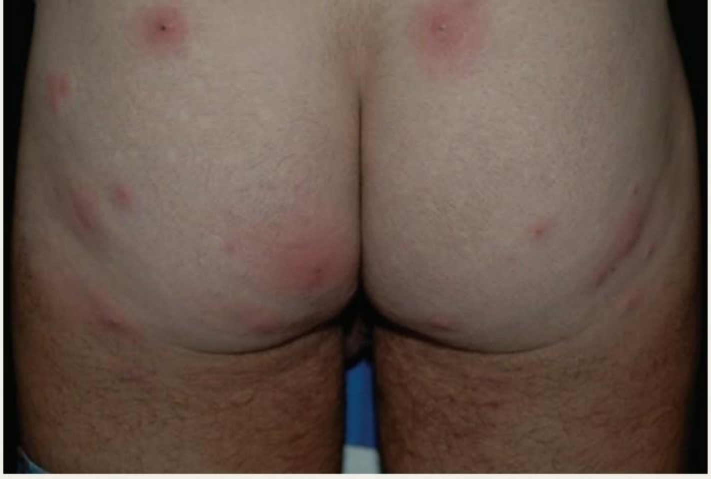


Figure 25-19. Multiple furuncles: MRSA Multiple painful nodules on the buttocks of a 44-year-old male with HIV disease.





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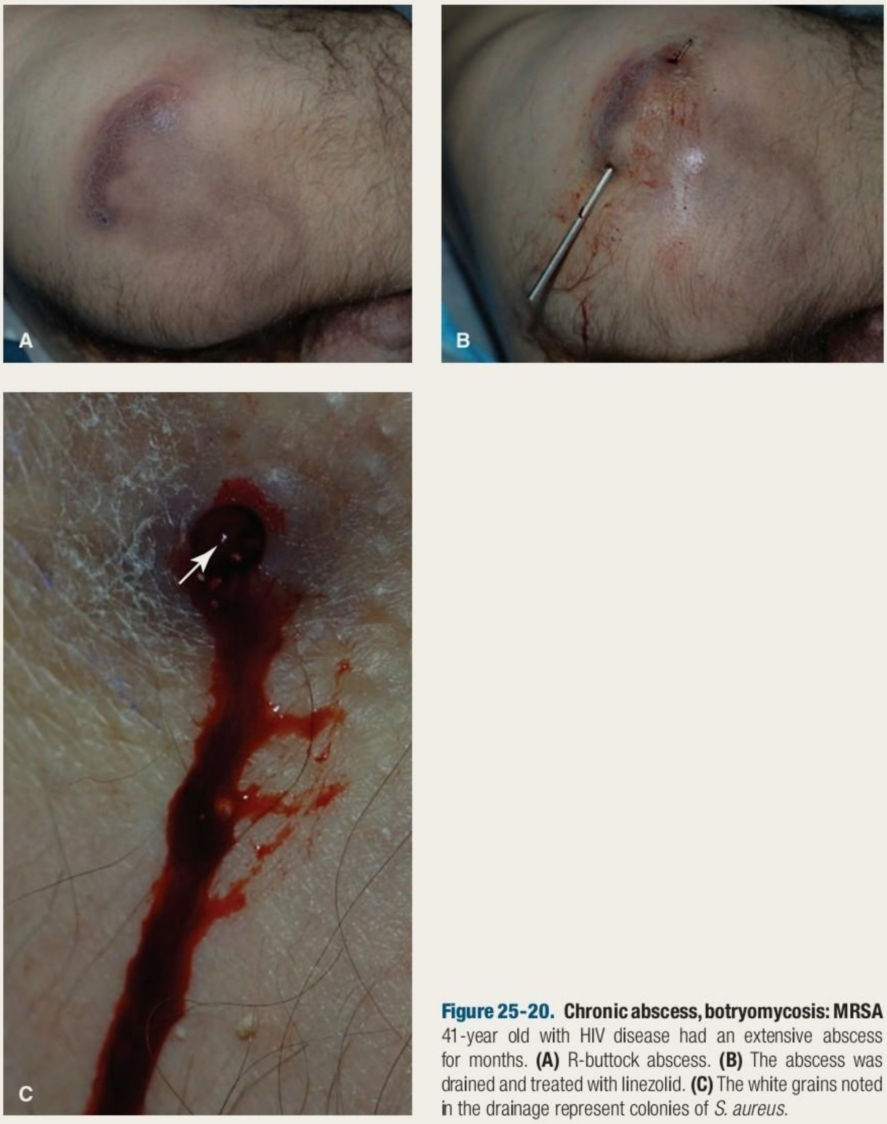

Figure 25-20. Chronic abscess, botryomycosis: MRSA 41-year old with HIV disease had an extensive abscess for months. **(A)** R-buttock abscess. **(B)** The abscess was drained and treated with linezolid. **(C)** The white grains noted in the drainage represent colonies of *S. aureus*.

drained and treated with linezolid. **(C)** The white grains noted in the drainage represent colonies of *S. aureus.*

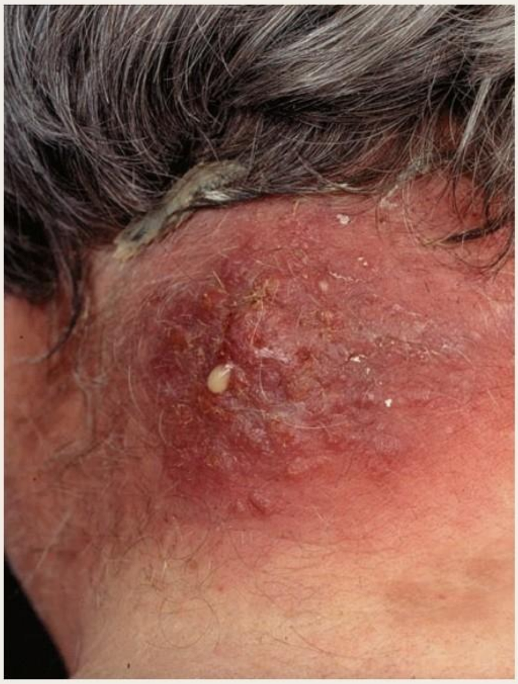

Figure 25-21. Carbuncle: MSSA A very large, inflammatory plaque studded with pustules, draining pus, on the nape of the neck. Infection extends down to the fascia and has formed from a confluence of many furuncles.

Soft-Tissue Infection

- Characterized by inflammation of skin and adjacent subcutaneous tissues. Soft tissue refers to tissues that connect, support, or surround other structures and organs: skin, adipose tissue, fibrous tissues, fascia, tendon, ligaments.
- **Syndromes.** Cellulitis, erysipelas, lymphangitis, necrotizing fasciitis, wound infection.
- **Soft-Tissue Inflammation.** Although often infectious, soft-tissue inflammation can be a manifestation of a noninfectious reaction pattern such as with neutrophilic dermatoses, erythema nodosum, and eosinophilic cellulitis.
- **Cellulitis.** Usually begins at a portal of entry in the skin, spreading proximally as an expanding solitary lesion. Uncommonly, soft-tissue infection can follow hematogenous dissemination with multiple sites of infection. Cellulitis is most often acute, caused by *S. aureus.*
- **Acute Inflammation.** Due to *cytokines* and bacterial *superantigens* rather than to overwhelming tissue infection.
- **Chronic Soft-Tissue Infection.** Nocardiosis, sporotrichosis, and phaeohyphomycosis.

Cellulitis ICD-9: 035 ∘ ICD-10: A46.0 ☐ ◐ → ○

- Acute, spreading infection of dermal and subcutaneous tissues. Characterized by a red, hot, tender area of skin. Portal of entry of infection is usually apparent. Most common pathogen is *S. aureus.* Erysipelas is a variant of cellulitis involving cutaneous lymphatics, and is usually caused by beta-hemolytic streptococci.

(Fig. 25-24). Lymphangitis. Lymph nodes can be enlarged and tender, regionally.

cial cutaneous lymphatic vessels, presenting a tender red expanding plaques, i.e., erysipelas

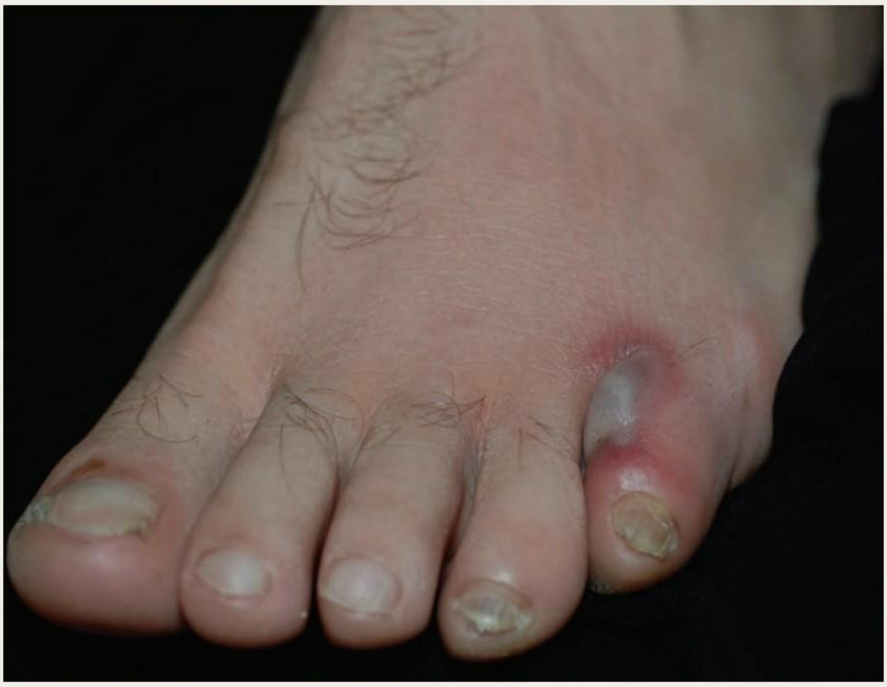

Figure 25-23. Cellulitis at portal of entry: MSSA 51-year-old male with interdigital tinea pedis noted pain on the dorsum of his foot. KOH preparation was positive for dermatophytic hyphae. Methicillin-sensitive *S. aureus* was isolated on culture of the webspace.

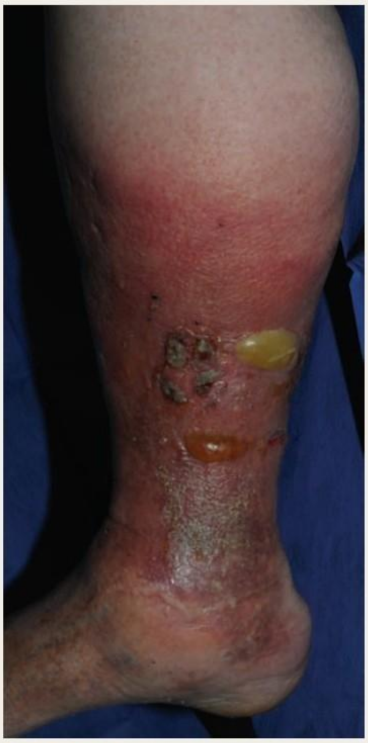

Figure 25-24. Cellulitis lower leg: MRSA 70-year-old obese male with chronic venous stasis and stasis ulcer had increasing erythema and blister formation of the lower leg associated with fever.

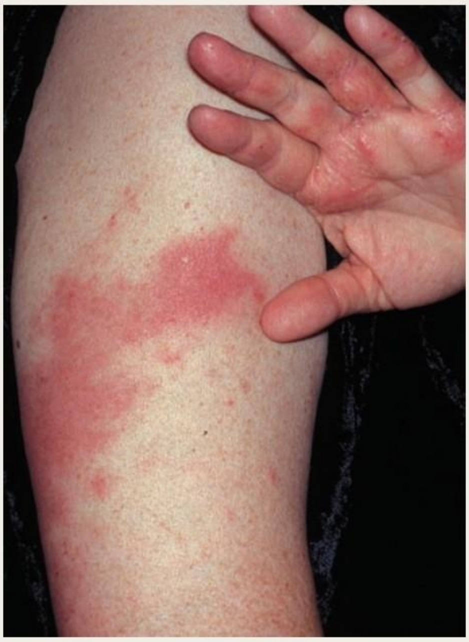

Figure 25-25. Recurrent cellulitis of the arm with chronic lymphedema: MSSA Right breast cancer had been treated with mastectomy and lymph node excision 10 years previously. Lymphedema of the right arm followed. Hand dermatitis was secondarily infected with MSSA. Cellulitis occurred repeatedly in the setting of chronic lymphedema.

(Fig. 25-27). Following childbirth, known as *puerperal sepsis*; infection can extend into pelvis. GBS cellulitis occurs in neonates; high morbidity and mortality. GAS infection with

center with erythematous halo, expanding rapidly without effective treatment (Fig. 25-29). *Distribution:* most commonly in the axilla, groin, perineum. Prognosis depends on prompt

E. rhusiopathiae: Erysipeloid occurs in individuals who handle game, poultry, fish. *Painful, inflamed plaque* with sharply defined irregular raised border occurring at the site of inoculation, i.e., finger or hand (Fig. 25-28), spreading to wrist and forearm. Color: purplish red acutely; brownish with resolution. Enlarges peripherally with central fading. Usually no systemic symptoms.

Ecthyma gangrenosum: Rare variant of necrotizing soft-tissue infection caused by *P. aeruginosa*. Clinically characterized by infarcted

a local infection in the absence of bacteremia, prognosis is much more favorable.

H. influenzae: Occurs mainly in children <2 years. Cheek, periorbital area, head, and neck are most common sites. Clinically, swelling, characteristic violaceous erythema hue. Use of Hib vaccine has dramatically reduced incidence.

V. vulnificus, *V. cholerae* non-01 and non-0139. Underlying disorders: cirrhosis, diabetes, immunosuppression, hemochromatosis, thalassemia. Follows ingestion of raw/undercooked seafood, gastroenteritis, bacteremia

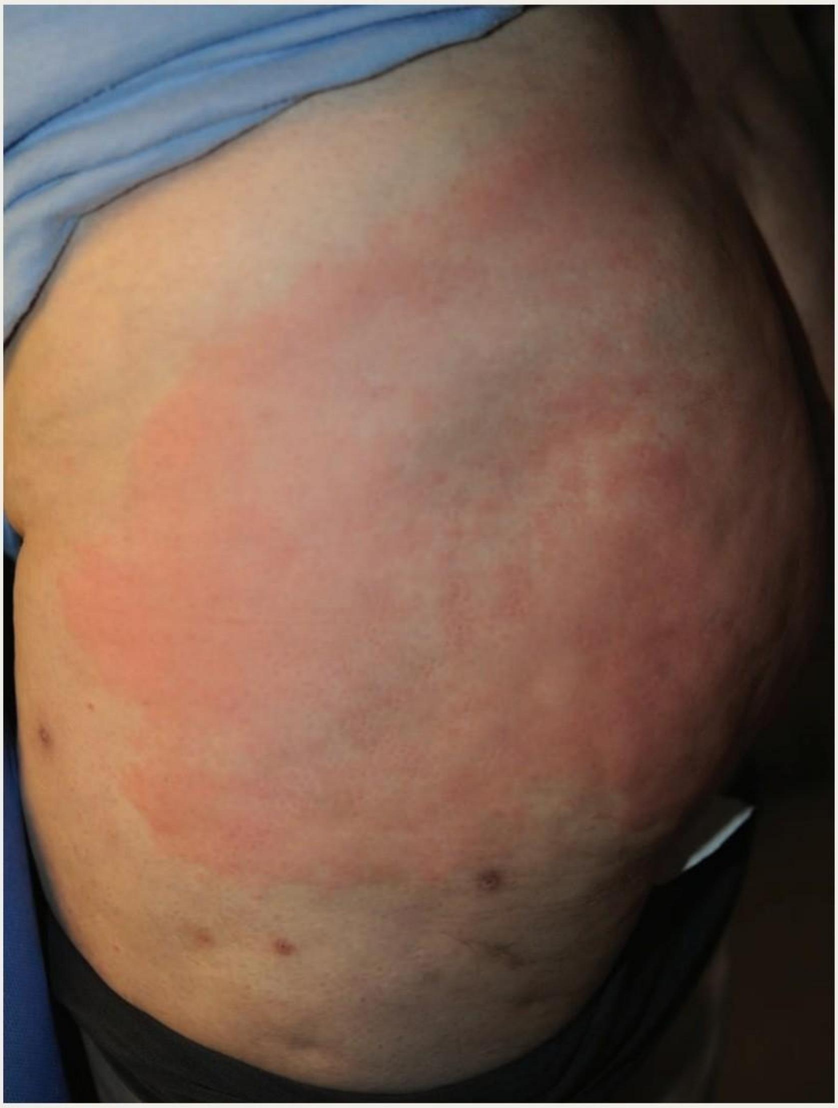

Figure 25-26. Erysipelas of buttocks: group B streptococcus 40-year-old female with history of Crohn disease with ileostomy, prior surgery for hidradenitis, and invasive vulvar carcinoma; treated with radiation. Portal of entry was intergluteal cleft. Presented with fever and local tenderness for 1 day.

with seeding of skin; also exposure of skin to seawater. Characterized by bulla formation, necrotizing vasculitis (Fig. 25-30). Usually on the extremities; often bilateral.

Aeromonas hydrophila: Water-associated trauma; preexisting wound. Immunocompromised host. Lower leg. Necrotizing soft-tissue infection.

C. canimorsus. Immunosuppression or asplenia; exposure to dog saliva or bite. Causes fulminant sepsis and disseminated intravascular coagulation (see Fig. 25-57).

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C. canimorsus. Immunosuppression or asplenia; exposure to dog saliva or bite. Causes fulminant sepsis and disseminated intravascular coagulation (see Fig. 25-57).

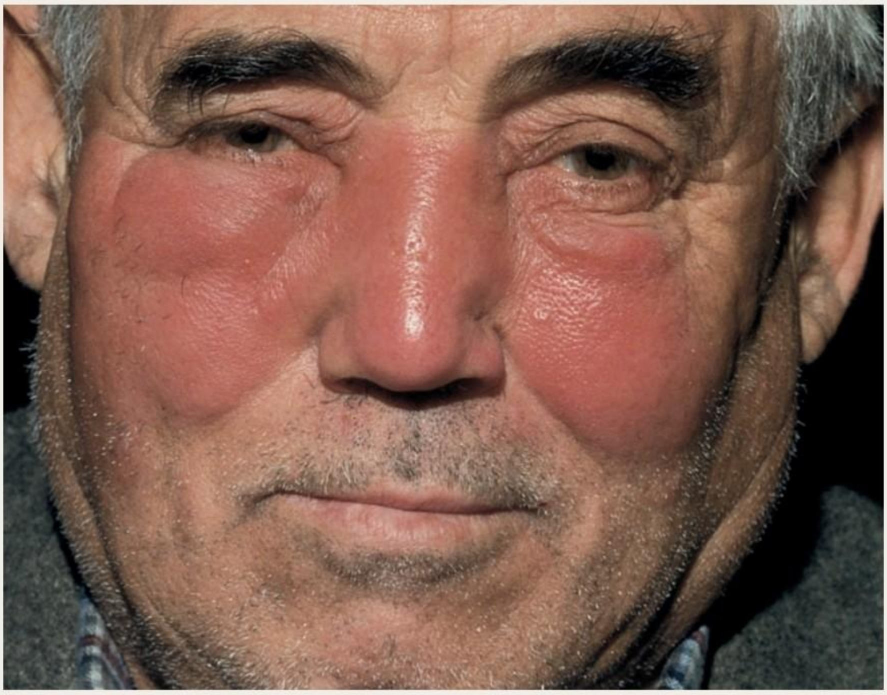
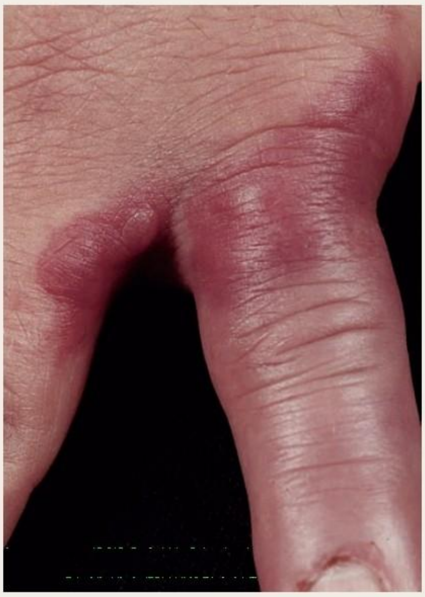


Figure 25-27. **Erysipelas of face: group A streptococcus** Painful, well-defined, shiny, erythematous, edematous plaques involving the central face of an otherwise healthy male. On palpation, the skin is hot and tender.

Figure 25-28. **Erysipeloid of hand** A well demarcated, violaceous, cellulitic plaque (without epidermal changes of scale or vesiculation) on the dorsa of the hand and fingers, occurred following cleaning fish; the site was somewhat painful, tender, and warm.



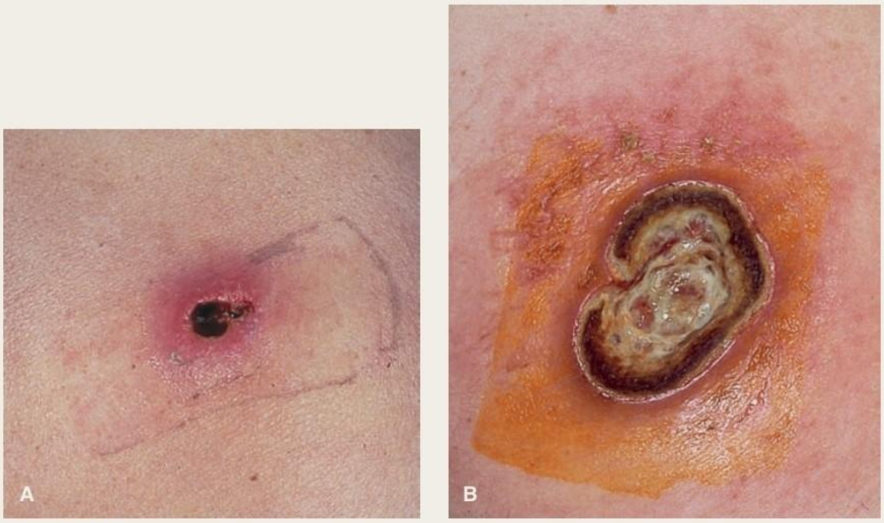


Figure 25-29. ***Ecthyma gangrenosum* of buttock: *P. aeruginosa*** A 30-year-old male with HIV disease and neutropenia. **(A)** An extremely painful, infarcted area with surrounding erythema present for 5 days. This primary cutaneous infection was associated with bacteremia. **(B)** Two weeks later, the lesion had progressed to a large ulceration. The patient died 3 months later of *P. aeruginosa pneumonitis* associated with chronic neutropenia.

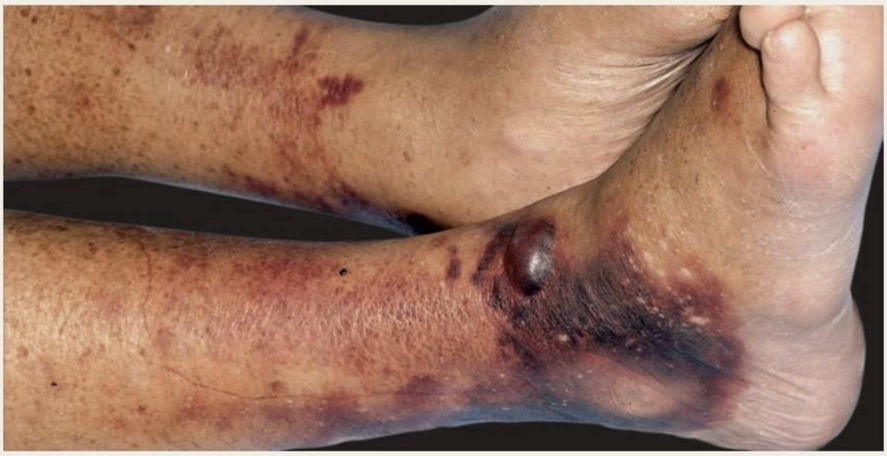


Figure 25-30. Bilateral cellulitis of legs: *V. vulnificus* Bilateral hemorrhagic plaques and bullae on the legs, ankles, and feet of an older diabetic with cirrhosis. Unlike other types of cellulitis in which microorganisms enter the skin locally, which is caused by *V. vulnificus*, usually follows a primary enteritis with bacteremia and dissemination to the skin. Most cases initially diagnosed as bilateral cellulitis are inflammatory (eczema, stasis dermatitis, psoriasis) rather than infectious.

production (crepitation on palpation), marked systemic toxicity. Necrotizing infection.

Nontuberculous mycobacteria (see p. 579).

Clinical diagnosis is based on morphologic features of lesion and the clinical setting, i.e., underlying diseases, travel history, animal often clustered in an intertriginous area.

Generalized Form. Exfoliative toxin-induced changes: *macular scarlatiniform rash* (staphylococcal scarlet fever syndrome) or diffuse, ill-defined erythema and a fine, stippled, sandpaper appearance occur initially. In 24 h, erythema deepens and involved skin becomes tender. Initially, periorificially on face, neck, axillae, groins; becoming more widespread in 24–48 h. Superficial epidermis is most pronounced periorificially on face; in flexural areas on neck, axillae, groins, antecubital areas; back (pressure points). With epidermolysis, epidermis appears wrinkled and can be removed by gentle pressure (skin resembles wet tissue paper) (*Nikolsky sign*) (Fig. 25-39). In some infants, flaccid bullae occur. Unroofed epidermis forms erosions with red, moist base

Differential Diagnosis

Kawasaki syndrome, adverse cutaneous drug eruption, scarlet fever.

Diagnosis

Clinical findings confirmed by bacterial cultures.

Course

With adequate antibiotic treatment, superficially denuded areas heal in 3–5 days associated with generalized desquamation; there is no scarring.

Treatment

Systemic antibiotic to treat infection and stop toxin production.

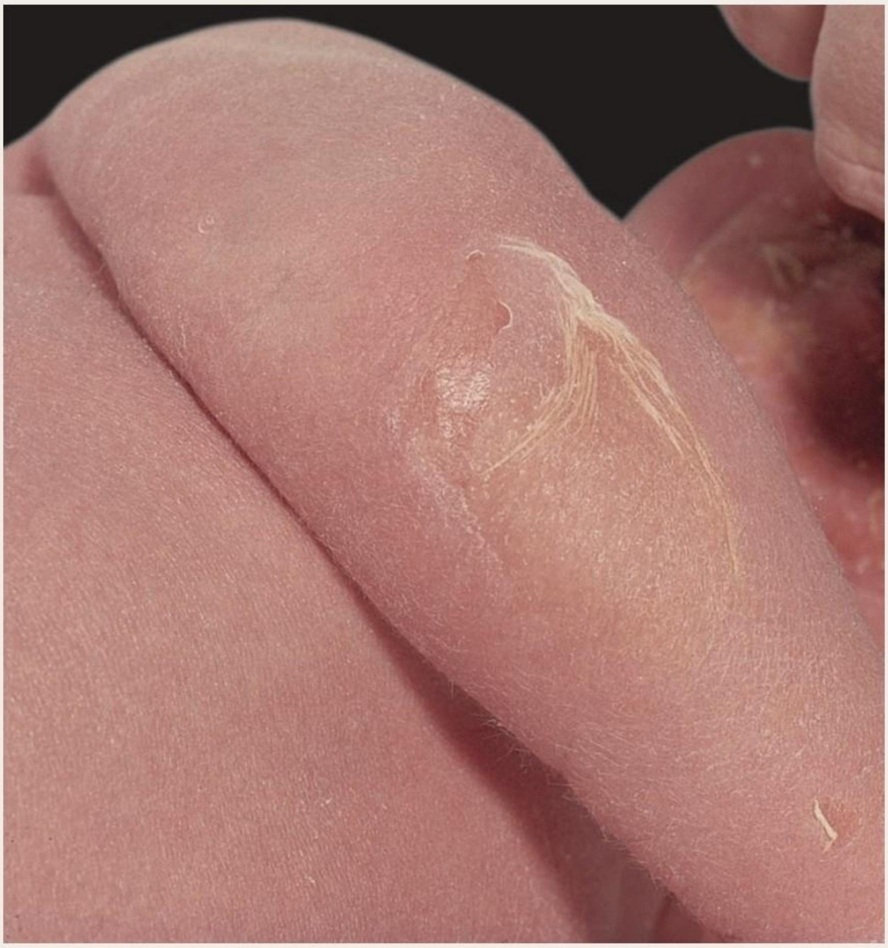


Figure 25-39. Staphylococcal scalded-skin syndrome: Nikolsky sign The skin of this infant is diffusely erythematous; gentle pressure to the skin of the arm has sheared off the epidermis, which folds like tissue paper.

Lepra Type 2 Reactions. Erythema nodosum leprosum (ENL). Seen in half of LL patients, usually occurring after initiation of antilepromatous therapy, generally within the first 2 years of treatment. Massive inflammation with erythema nodosum–like lesions.

Lucio Reaction. Individuals with diffuse LL develop shallow, large polygonal sloughing ulcerations on the legs. The reaction appears to be either a variant of ENL or secondary to arteriolar occlusion.

Clinical Manifestation

Incubation period is 2–40 years (most commonly 5–7 years). Onset is insidious and painless; first affects peripheral nervous system with persistent or recurrent painful paresthesias and numbness without any visible clinical signs. At this stage, there may be transient macular skin eruptions; blister, but lack of awareness of trauma. Neural involvement leads to muscle weakness, muscle atrophy, severe neuritic pain, and contractures of the hands and feet.

Tuberculoid Leprosy (TT, BT). Few well-defined *hypopigmented hypesthetic macules* (Fig. 25-62) with raised edges and varying in size from a few millimeters to very large lesions covering the entire trunk. Erythematous or purple border and hypopigmented center. Sharply defined, raised; often annular; enlarge peripherally. Central area becomes atrophic or depressed. Advanced lesions are anesthetic, devoid of skin appendages (sweat glands, hair follicles). Any site including the face. *TT*: Lesions may resolve spontaneously; not associated with lepra reactions. *BT*: Does not heal spontaneously; type 1 lepra reactions may occur.

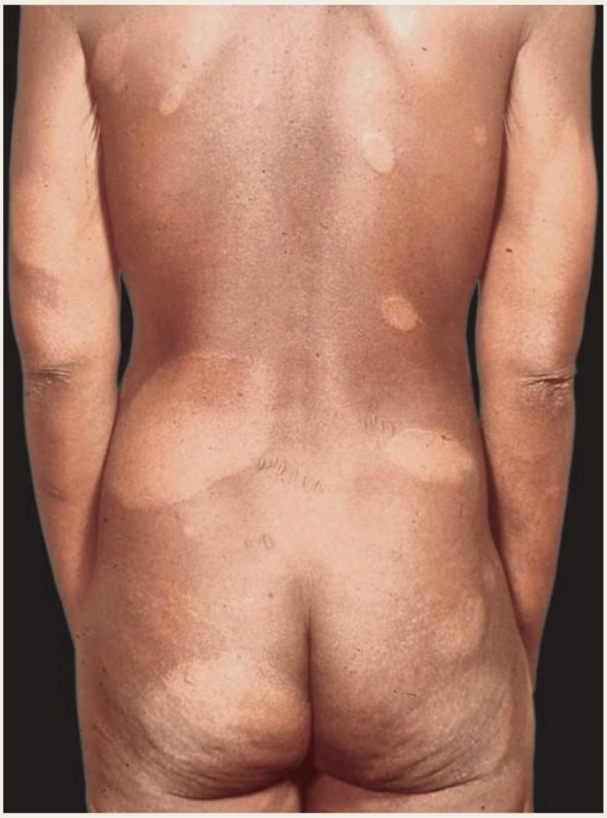


Figure 25-62. Leprosy: tuberculoid type Well-defined, hypopigmented, slightly scaling, anesthetic macules and plaques on the posterior trunk.

Nerve Involvement: May be a thickened nerve on the edge of the lesion; large peripheral nerve enlargement frequent (ulnar, posterior auricular, peroneal, posterior tibial nerves). Skin involvement is absent in *neural leprosy.* Nerve involvement associated with hypesthesia (pinprick, temperature, vibration) and myopathy.

Borderline BB Leprosy. Lesions are intermediate between tuberculoid and lepromatous and are composed of macules, papules, and plaques (Fig. 25-63). Anesthesia and decreased sweating are prominent in the lesions.

Lepromatous Leprosy (LL, BL). Skin-colored or slightly erythematous papules/nodules. Lesions enlarge; new lesions occur and coalesce. Later: symmetrically distributed nodules, raised plaques, diffuse dermal infiltrate, which on face results in loss of hair (lateral eyebrows and eyelashes) and leonine facies (lion's face; Fig. 25-64). *Diffuse lepromatosis,* occurring in western Mexico, Caribbean, presents as diffuse dermal infiltration and thickened dermis. Bilaterally symmetric involving earlobes, face, arms, and buttocks, or less frequently the trunk and lower extremities. Tongue: nodules, plaques, or fissures.

Nerve Involvement: More extensive than in TT.

Other Involvement: Upper respiratory tract, anterior chamber of eye, testes.

Reactional States

Immunologically mediated inflammatory states, occurring spontaneously or after initiation of therapy.

Lepra Type 1 Reactions: Skin lesions become acutely inflamed, associated with edema and pain; may ulcerate. Edema most severe on face, hands, and feet.

Lepra Type 2 Reactions (ENL): Present as painful red skin nodules arising superficially and deeply, in contrast to true erythema nodosum. Lesions form abscesses or ulcerate; occur most commonly on face and extensor limbs.

Lucio Reaction: Occurs only in patients from Mexico or Caribbean with diffuse LL. Presents face results in loss of hair (lateral eyebrows and eyelashes) and leonine facies (lion's face; Fig. 25-64). *Diffuse lepromatosis*, occurring in western Mexico, Caribbean, presents as diffuse dermal infiltration and thickened dermis.

nodosum. Lesions form abscesses or ulcerate, occur most commonly on face and extensor limbs.

Lucio Reaction: Occurs only in patients from Mexico or Caribbean with diffuse LL. Presents

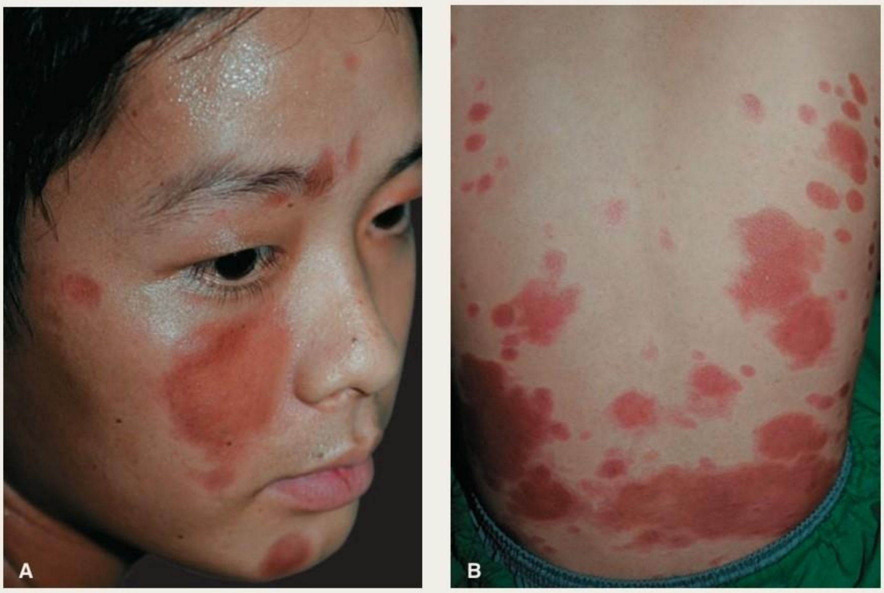


Figure 25-63. Leprosy: borderline-type A 26-year-old Vietnamese male. **(A)** Well-demarcated, infiltrated, erythematous plaques on the face. **(B)** Identical red plaques on the lower back.

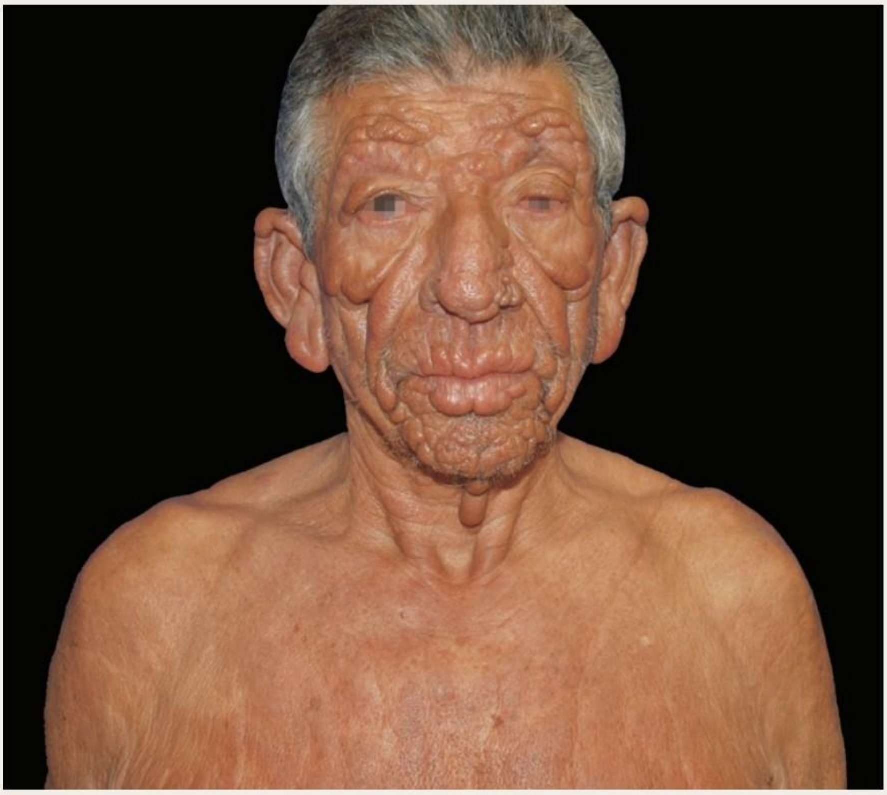


Figure 25-64. Diffuse skin infiltration, multiple nodular lesions, and sensory loss are the key hallmarks of lepromatous leprosy (LL). This patient presented lesions on the upper part of the thorax, forehead, ears, nose, lips, perilabial, and mentonian regions, as well as lax skin of the malar and palpebral superior regions, with muscle force impairment on the left side. Superciliary and ciliary madarosis were also present. Ulnar and tibial posterior nerves were enlarged. A Ziehl–Neelsen stained skin smear had a 6+ bacterial index for acid-fast bacilli in clumps, and ELISA titration for anti-PGL-1 IgM was 3.445 (cutoff 0.295). The 12-month World Health Organization multidrug therapy regimen and prednisone were prescribed, with significant improvement. LL is the anergic form of leprosy; it generates an exacerbated but inefficient humoral immune response, leading to highly infectious patients. Mycosis fungoides, neurofibromatosis, sarcoidosis, amyloidosis, syphilis, anergic leishmaniasis, and lobomycosis are among diseases in the differential diagnosis. (Courtesy of C. G. Salgado and J. G. Barreto, Pará Federal University, Brazil.)

loidosis, syphilis, anergic leishmaniasis, and lobomycosis are among diseases in the differential diagnosis. (Courtesy of C. G. Salgado and J. G. Barreto, Pará Federal University, Brazil.)

as irregularly shaped erythematous plaques; lesions may resolve spontaneously or undergo necrosis with ulceration.

General Findings

Extremities: Sensory neuropathy, plantar ulcers, secondary infection; ulnar and peroneal palsies (Fig. 25-65), Charcot joints. Squamous cell carcinoma can arise in chronic foot ulcers (Fig. 11-13).

Nose: Chronic nasal congestion, epistaxis; destruction of cartilage with saddle-nose deformity (Fig. 25-63).

Eyes: Cranial nerve palsies, lagophthalmus, corneal insensitivity. In LL, anterior chamber can be invaded with uveitis, glaucoma, cataract formation. Corneal damage can occur secondary to trichiasis and sensory neuropathy, secondary infection, and muscle paralysis.

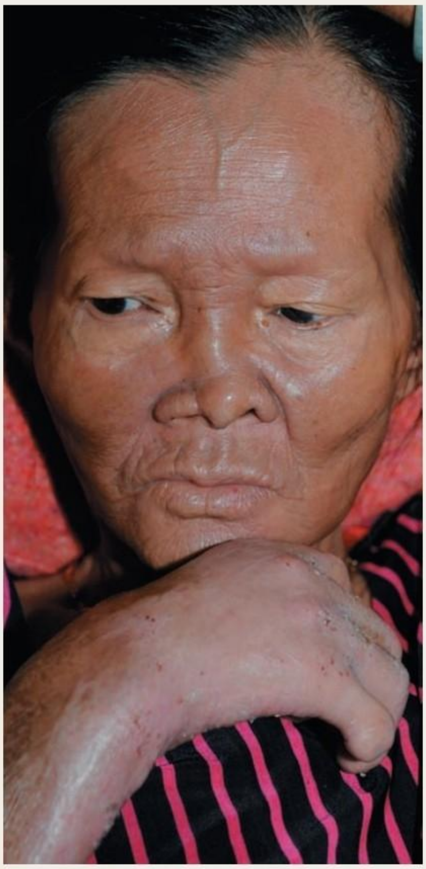


Figure 25-65. Leprosy: lepromatous type A 60-year-old Vietnamese female with treated advanced disease. Ulnar palsy, loss of digits on right hand, and saddle-nose deformity associated with loss of nasal cartilage are seen.

Testes: May be involved in LL with resultant hypogonadism.

Complications of Leprosy: *Squamous cell carcinoma* can arise in chronic neurotrophic ulcers on the lower extremities (see Fig. 11-13). The tumors are usually low-grade malignancies but can metastasize to regional lymph nodes and cause death. *Secondary amyloidosis* with hepatic and renal abnormalities.

Differential Diagnosis

Hypopigmented lesions with granulomas.

Sarcoidosis, leishmaniasis, NTM infection, lymphoma, syphilis, granuloma annulare.

Laboratory Examinations

Slit-Skin Smears. A small skin incision is made; the site is then scraped to obtain tissue fluid from which a smear is made and examined after Ziehl–Neelsen staining. Specimens are usually obtained from both earlobes and two other active lesions. Negative BIs are seen in paucibacillary cases, treated cases, and cases examined by an inexperienced technician.

Culture. *M. leprae* has not been cultured in vitro; however, it does grow when inoculated into the mouse foot pad. Routine bacterial cultures to rule out secondary infection.

PCR. *M. leprae* DNA detected by this technique makes the diagnosis of early paucibacillary leprosy and identifies *M. leprae* after therapy.

Serology. Measure IgM antibodies to phenolic glycolipid-1 (PGL-1).

Dermatopathology. TL shows epithelioid cell granulomas forming around dermal nerves; AFB are sparse or absent. LL shows an extensive cellular infiltrate separated from the epidermis by a narrow zone of normal collagen. Skin appendages are destroyed. Macrophages are filled with *M. leprae,* having abundant foamy or vacuolated cytoplasm (lepra cells or Virchow cells).

Diagnosis

Made if one or more of the cardinal findings are detected: patient from endemic area, skin lesions characteristic of leprosy with diminished or loss of sensation, enlarged peripheral nerves, finding of *M. leprae* in skin or, less commonly, other sites.

Course

After the first few years of drug therapy, the most difficult problem is management of the changes secondary to neurologic deficits—contractures and trophic changes in the hands and feet. Uncommonly, secondary amyloidosis with renal failure can complicate long-standing leprosy. Lepra type 1 reactions last 2–4 months in individuals with BT and up to 9 months in those with BL. Lepra type 2 reactions (ENL) occur in 50% of individuals with LL and 25% of those with BL within the first 2 years of treatment. ENL may be complicated by uveitis, dactylitis, arthritis, neuritis, lymphadenitis, myositis, orchitis. Lucio reaction or phenomenon occurs secondary to vasculitis with subsequent infarction.

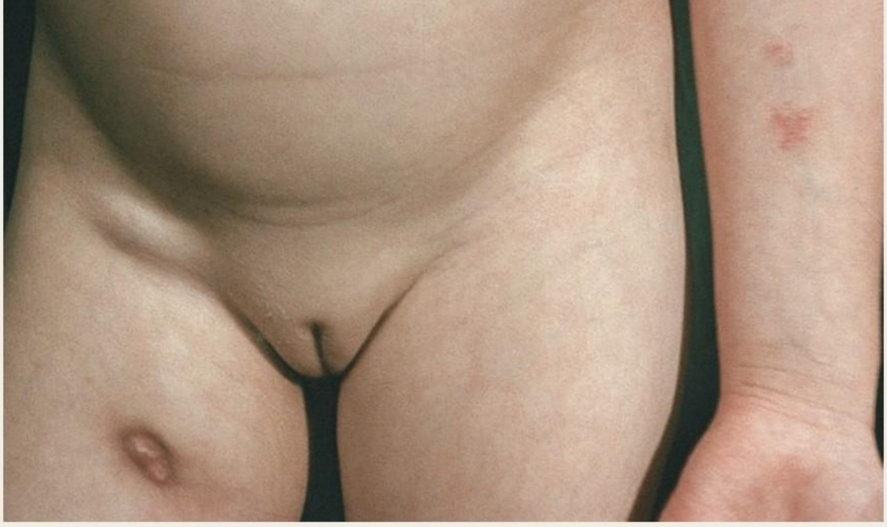
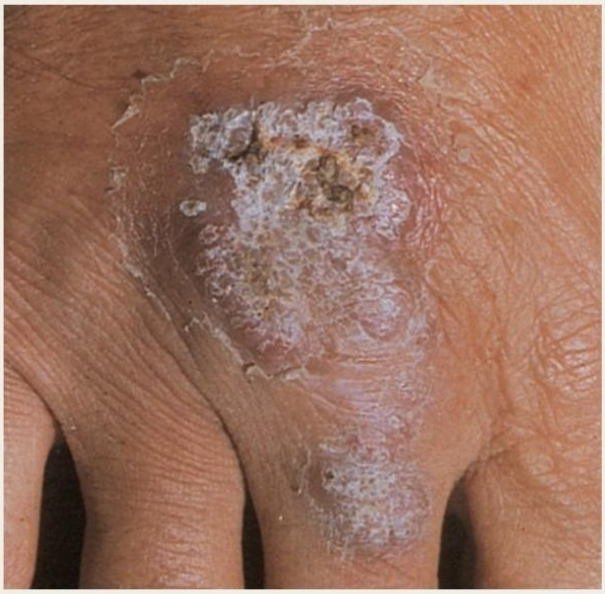


Figure 25-66. Primary inoculation tuberculosis A large, ulcerated nodule at the site of *Mycobacterium* tuberculosis inoculation on the right thigh associated with inguinal lymphadenopathy. The erythematous papules on the left forearm occurred at the site of tuberculin testing.

Figure 25-67. Tuberculosis verrucosa cutis A 40-year-old male with warty and crusted plaques on the dorsum of the R-hand for 6 months. [From Sethi A. Tuberculosis and infections with atypical *Mycobacteria*. In Goldsmith LA et al. (eds.). *Fitzpatrick's Dermatology in General Medicine*, 8th ed. New York, McGraw-Hill, 2012.]



Lupus Vulgaris. Initial papule ill defined and soft and evolves into *well-defined, irregular plaque* (Fig. 25-68). Reddish-brown. Diascopy (glass slide pressed against skin) shows semitranslucent *"apple jelly" color* (i.e., orange-tan). Lesions are characteristically soft and friable. Surface is initially smooth or slightly scaly but may become hyperkeratotic. Hypertrophic forms result in soft tumorous nodules. Ulcerative forms present as punched-out, often serpiginous ulcers surrounded by soft, brownish infiltrate. Usually solitary, but several sites may occur. *Most lesions on the head and neck*, most often on nose, ears, or scalp. Lesions on ears or nose can result in destruction of underlying cartilage. *Scarring is prominent.* Characteristically new brownish infiltrates occur within atrophic scars.

Scrofuloderma. Firm subcutaneous nodule that initially is freely movable; lesion then becomes doughy and evolves into irregular, *deep-seated node or plaque* that liquefies and perforates (Fig. 25-69). Ulcers and irregular sinuses, usually of linear or serpiginous shape, discharge pus or caseous material. Edges are undermined, inverted, with dissecting subcutaneous pockets alternating with soft, fluctuating infiltrates and bridging scars. Most often occurs in the *parotid, submandibular*, and *supraclavicular regions*; lateral neck; scrofuloderma most often results from

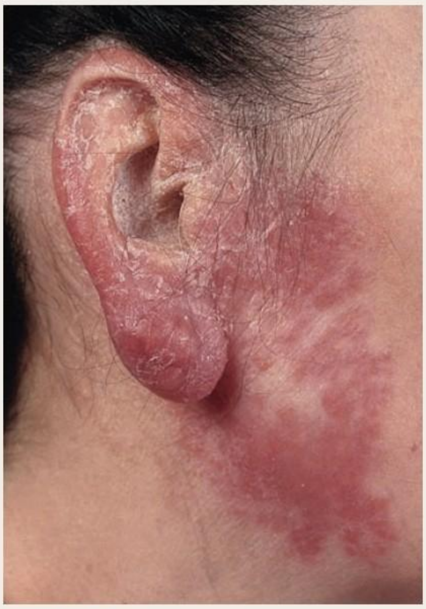


Figure 25-68. Lupus vulgaris Reddish-brown plaque, which on diascopy exhibits the diagnostic yellow-brown apple-jelly color. Note nodular infiltration of the earlobe, scaling of the helix, and atrophic scarring in the center of the plaque.

Scrofuloderma. Firm subcutaneous nodule that initially is freely movable; lesion then becomes doughy and evolves into irregular, *deep-seated node or plaque* that liquefies and perforates (Fig. 25-69). Ulcers and irregular sinuses, usually of linear or serpiginous shape, discharge pus or caseous material. Edges are undermined, inverted, with dissecting subcutaneous pockets alternating with soft, fluctuating infiltrates and bridging scars. Most often occurs in the *parotid*, *submandibular*, and *supraclavicular regions*; lateral neck; scrofuloderma most often results from contiguous spread from affected lymph nodes or tuberculous bones (phalanges, sternum, ribs) or joints.

Figure 25-68. Lupus vulgaris Reddish-brown plaque, which on diascopy exhibits the diagnostic yellow-brown apple-jelly color. Note nodular infiltration of the earlobe, scaling of the helix, and atrophic scarring in the center of the plaque.

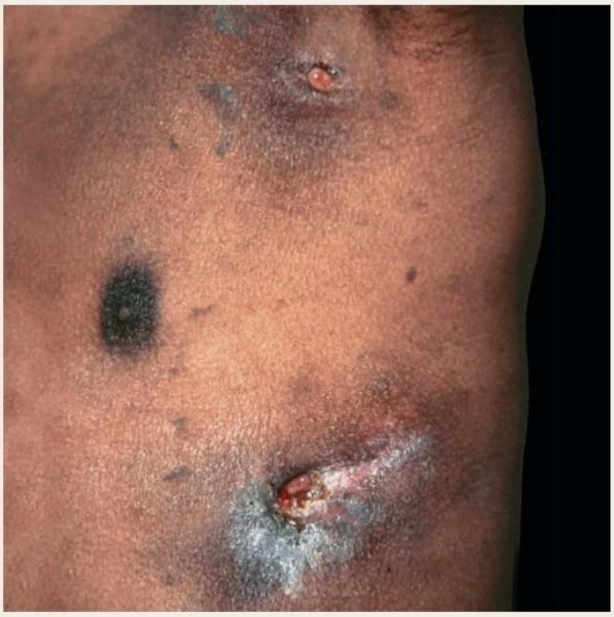


Figure 25-69. Scrofuloderma: lateral chest wall. Two ulcers on the chest wall and axilla are associated with underlying sinus tracts.

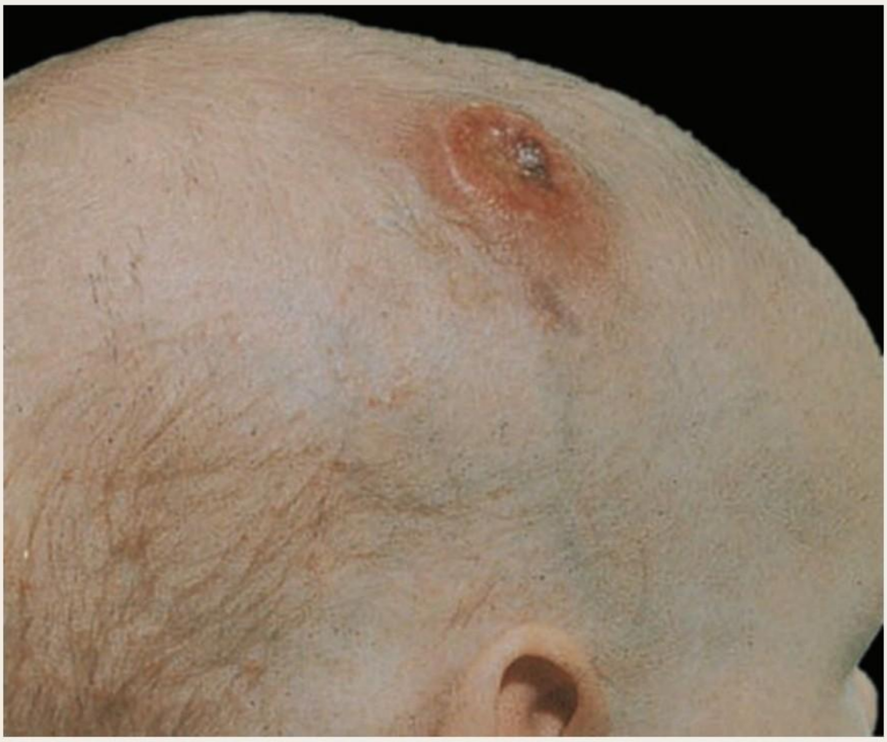


Figure 25-70. Metastatic tuberculous abscess on the scalp An infant with combined immunodeficiency. Note abscess formation and discharge of purulent material but little inflammation.

Metastatic Tuberculosis Abscess. Subcutaneous abscess, nontender, "cold," fluctuant. Co-

from progressive tuberculosis of internal organs, it is usually found on the oral, pharyngeal (pulmonary tuberculosis), vulvar (genitourinary tuberculosis), and anal (intestinal tuberculosis) mucous membranes. Lesions may be or purpuric lesions. Sometimes vesicular and crusted. Removal of crust reveals umbilication. Disseminated on all parts of body, particularly trunk.

Orificial Tuberculosis. Small yellowish nodule on mucosa breaks down to form *painful circular or irregular ulcer* (Fig. 25-71) with undermined borders. Surrounding mucosa swollen, edematous, and inflamed. Since orifical tuberculosis results from autoinoculation of mycobacteria

Clinical findings, tuberculin skin testing (Fig. 25-72), dermatopathology, confirmed by isolation of *M. tuberculosis* on culture or by PCR.

Course

The course of cutaneous tuberculosis is quite variable, depending on the type of cutaneous infection, amount of inoculum, extent of extracutaneous infection, age of the patient, immune status, and therapy.

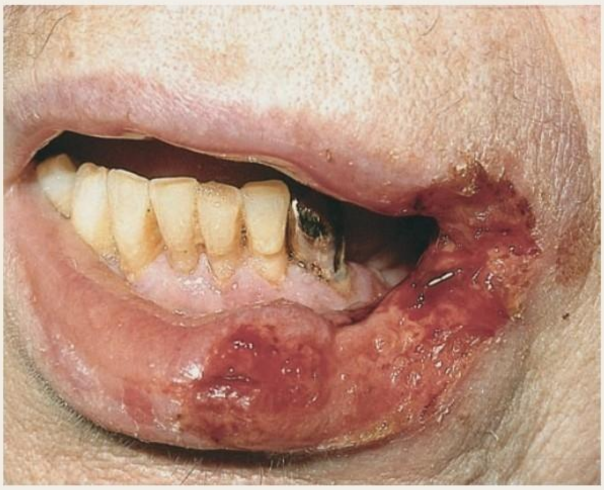


Figure 25-71. Orificial tuberculosis: lips A large, very painful ulcer on the lips of this patient with advanced cavitary pulmonary tuberculosis.

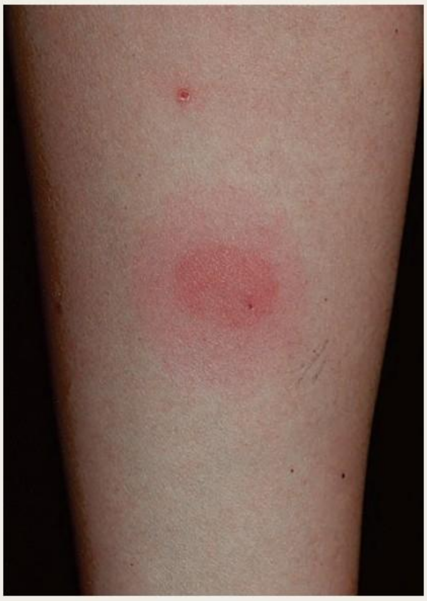


Figure 25-72. Purified protein derivative or Mantoux test: positive test A 31-year-old Taiwanese female with psoriasis, with a negative skin test 1 year previously, was retested prior to beginning etanercept. She had become infected while visiting her father, who had pulmonary tuberculosis, in Taiwan. A red plaque with surrounding erythema is seen at the test site.

Treatment

Only PIT and TVC are limited to the skin. All other patterns of cutaneous tuberculosis are associated with systemic infection that has disseminated secondarily to skin. As such, therapy should be aimed at achieving a cure, avoiding relapse, and preventing emergence of drug-resistant mutants.

Antituberculous Therapy. Prolonged antituberculous therapy with at least two drugs is indicated for all cases of CTb except for TVC that can be excised.

- Standard antituberculous therapy:
  - Isoniazid (5 mg/kg daily) plus
  - Rifampin (600 mg/kg daily)
- Supplemented in initial phases with:
  - Ethambutol (25 mg/kg daily) and/or
  - Streptomycin (10–15 mg/kg daily) and/or
  - Pyrazinamide (15–30 mg/kg daily)

Isoniazid and rifampin for at least 9 months; can be shortened to 6 months if four drugs are given during the first 2 months.

Multidrug Resistant (MDR) Tb. Incidence is increasing.

Nontuberculous Mycobacterial Infections

Interdigital. Most common in obese elderly. Initial pustule becomes eroded, with formation of superficial erosion or fissure (Fig. 26-4). May be associated with *Candida* paronychia.

Occluded Skin. Under occlusive dressing, under cast, on back in hospitalized patient.

Follicular Candidiasis. Small, discrete pustules in ostia of hair follicles. Usually in occluded skin.

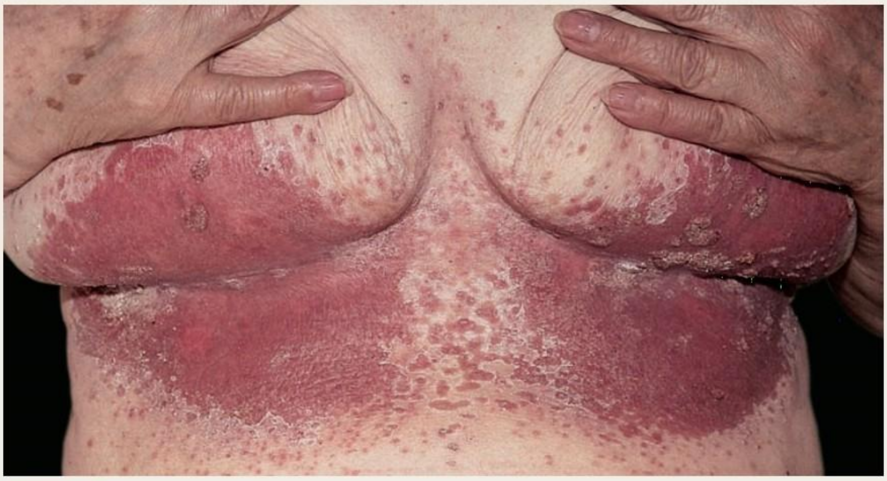


Figure 26-2. Cutaneous candidiasis: intertrigo Small peripheral "satellite" papules and pustules that have become confluent centrally, creating a large eroded area in the submammary region.

Differential Diagnosis

Intertrigo/Occluded Skin. Intertriginous psoriasis, erythrasma, dermatophytosis, pityriasis versicolor, streptococcal intertrigo, **Diaper Dermatitis.** Atopic dermatitis, psoriasis, irritant dermatitis, seborrheic dermatitis.

Folliculitis. Bacterial (*Staphylococcus aureus, Pseudomonas aeruginosa*) folliculitis, *Pityrosporum* folliculitis, acne.

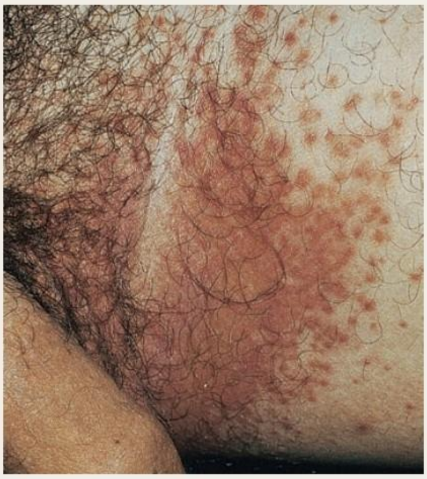


Figure 26-3. Cutaneous candidiasis: intertrigo Erythematous papules with a few pustules, becoming confluent in the inguinal area and medial thigh. The lesions occurred during a holiday trip to the Caribbean.

Diagnosis

Clinical findings confirmed by direct microscopy or culture.

Treatment

Prevention. Keep intertriginous areas dry, wash with benzoyl peroxide bar and use imidazole powder.

Topical Antifungals. Nystatin, azole, or imidazole cream.

Oral Antifungals. Nystatin (suspension, tablet, pastille.) Eradicates bowel colonization. May be effective in recurrent candidiasis of diaper area, genitals, or intertrigo.

Systemic Antifungal Agents. Fluconazole tablets (50, 100, 150, 200 mg), oral suspension (50 mg/ml); parenteral for IV infusion. Itroconazole capsules (100 mg), oral solution 10 mg/ml), ketoconazole tablets (200 mg), amphotericin B IV for severe disease.

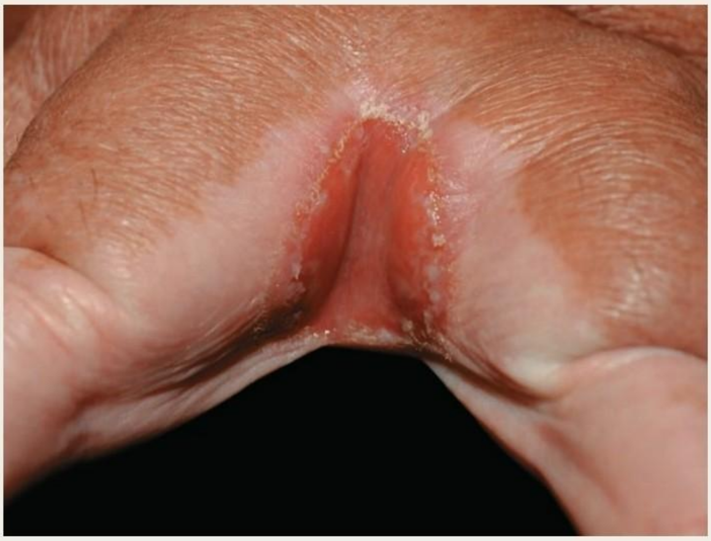


Figure 26-4. Cutaneous candidiasis: interdigital intertrigo An 80-year-old male with painful site in the webspace of the hand. Erosion with erythema is seen in the webspace between two fingers.

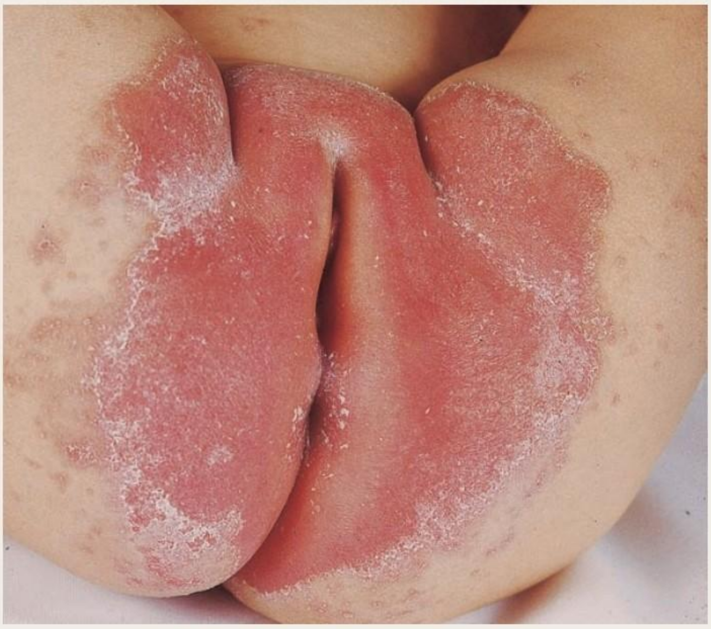


Figure 26-5. Candidiasis: diaper dermatitis Confluent erosions, marginal scaling, and "satellite pustules" in the area covered by a diaper in an infant. Atopic dermatitis or psoriasis also occurs in this distribution and may be concurrent.

Oropharyngeal Candidiasis
ICD-9: 112.0 ∘ ICD-10: B38.0

◧ ◐

- Occurs with minor variations in host factors. Antibiotic therapy; glucocorticoid therapy (topical or systemic); age (very young, very old); host defense defects.

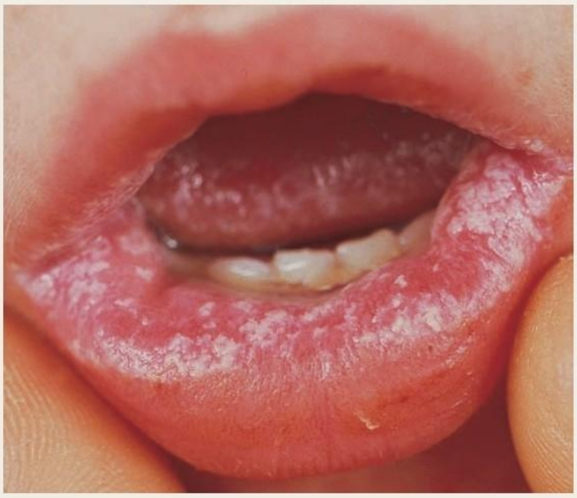


Figure 26-6. Oral candidiasis: thrush White curd-like material on the mucosal surface of the lower lip of a child; the material can be abraded with gauze (pseudomembranous), revealing underlying erythema.

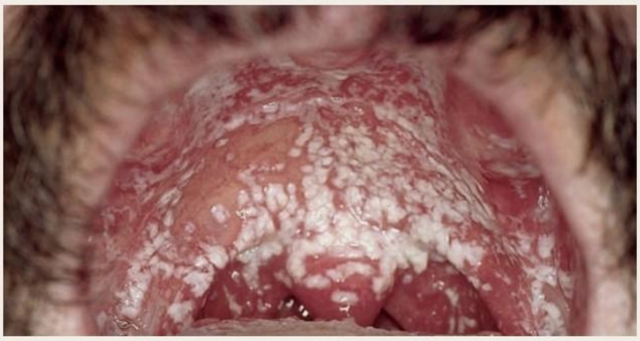


Figure 26-7. Oral candidiasis: thrush Extensive cottage cheese-like plaques, colonies of *Candida* that can be removed by rubbing with gauze (pseudomembranous), on the palate and uvula of an individual with advanced HIV/AIDS. Patches of erythema between the white plaques represent erythematous (atrophic) candidiasis. Involvement may extend into the esophagus and become associated with dysphagia.

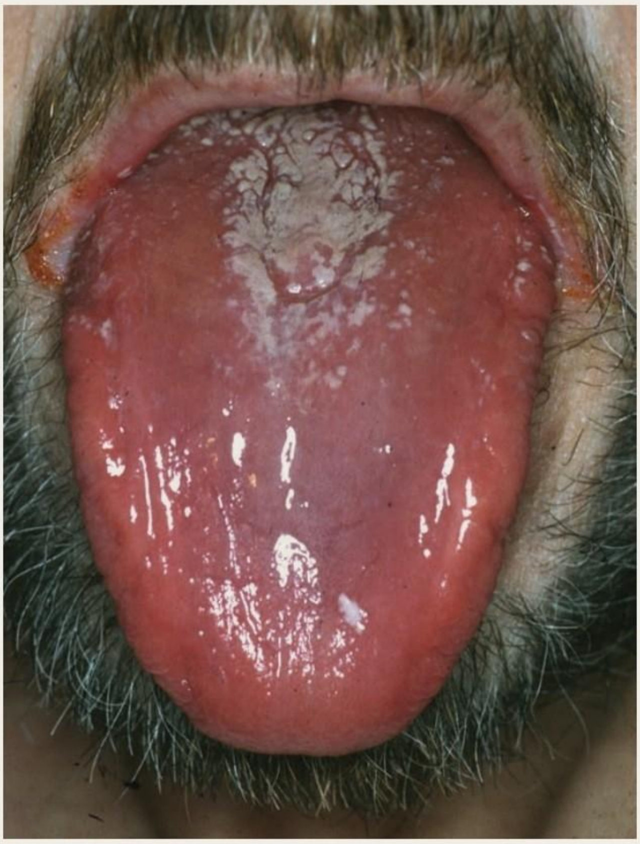
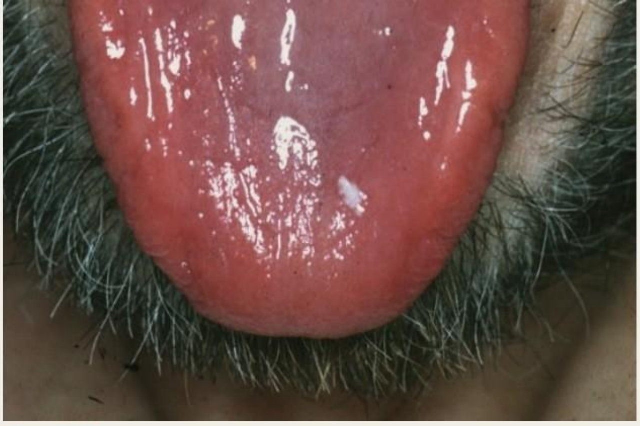


Figure 26-8. Oral candidiasis: atrophic and pseudomembranous A 48-year-old male with HIV disease. The surface of the tongue is shiny and red; posterior tongue has a white coating (thrush).



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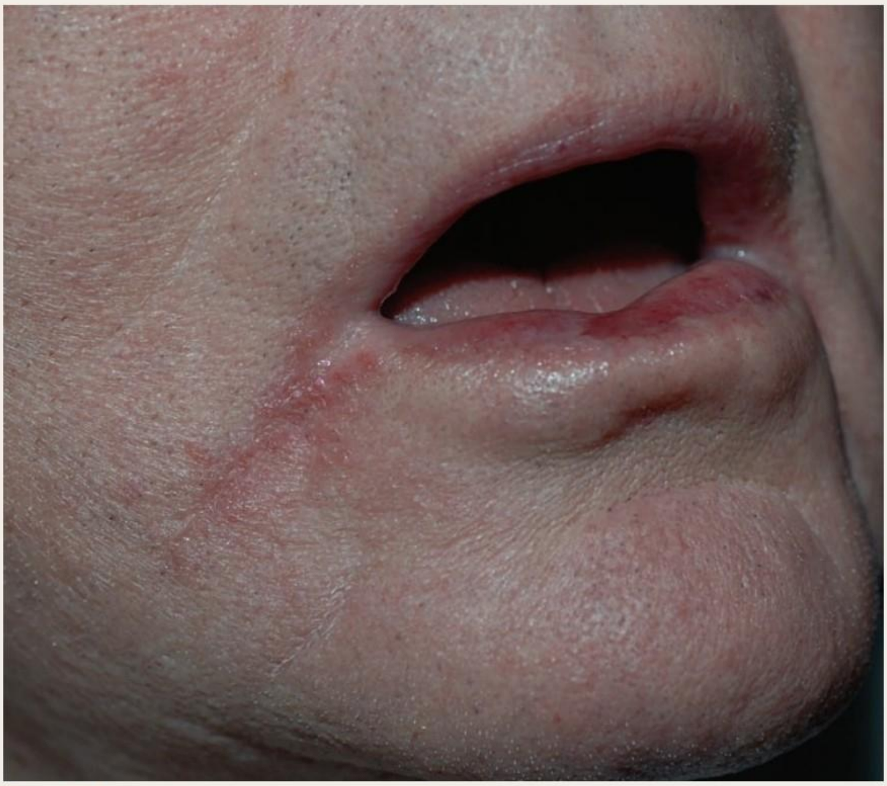

Figure 26-9. Angular cheilitis A 55-year-old male. The angle of the lips is moist and red. KOH preparation revealed candida pseudohyphae. Oral candidiasis was also present.

Esophageal and Tracheobronchial Candidiasis. Occurs in HIV disease when CD4+ cell count is low and is an AIDS-defining condition. Odynophagia, resulting in difficulty eating and malnutrition. Pseudomembranous lesions are seen on endoscopy.

Invasive Disseminated Candidiasis. In individuals with severe prolonged neutropenia. Portal of entry of *Candida*: GI tract, invading submucosa, and blood vessels; intravascular catheter. Candidemia: hematogenous dissemination to skin and viscera. Disseminated red papules (Fig. 26-14)

Differential Diagnosis

Pseudomembranous Candidiasis. Oral hairy leukoplakia, condyloma acuminatum, geographic tongue, hairy tongue, lichen planus, bite irritation.

Atrophic Candidiasis. Lichen planus, poor nutrition, vitamin deficiency

Diagnosis

Clinical suspicion confirmed by KOH preparation of scraping from mucosal surface. Endoscopy to document esophageal and/or tracheobronchial candidiasis.

Course

Most cases respond to correction of the precipitating cause such as use of inhaled glucocorticoids. Topical agents effective in most cases. Clinical resistance to antifungal agents may be related to patient noncompliance, severe immunocompromised, drug–drug interaction (rifampin–fluconazole).

Treatment

Topical Therapy. Nystatin or clotrimazole.

Systemic Therapy. Oral fluconazole, itraconazole, ketoconazole. Amphotericin B for severe resistant disease.

80–90% of genital isolates.

Incidence. Most vaginal candidiasis occurs in the healthy population. Seventy-five percent of women experience at least one episode; 40–45% experience two or more episodes. Often associated with vulvar candidiasis, i.e., vulvovaginal candidiasis.

Risk Factors. Diabetes mellitus, HIV disease. Females: Often none; pregnancy. Males; uncircumcised.

Clinical Manifestation

Vulvitis/Vulvovaginitis. Onset often abrupt, usually the week before menstruation. Symptoms may recur before each menstruation. Pruritus, vaginal discharge, vaginal soreness, vulvar burning, dyspareunia, and external dysuria.

Vulvitis. Erosions, edema, erythema (Fig. 26-10), swelling, removable curd-like material. Pustule on lateral vulva and adjacent skin

Vulvovaginitis. Vaginal erythema and edema; white plaques that can be wiped off vaginal and/or cervical mucosa. May be associated with candidal intertrigo of inguinal folds and perineum. Subcorneal pustules at periphery with fringed, irregular margins. In chronic cases, vaginal mucosa glazed and atrophic.

Balanoposthitis, balanitis glans, and preputial sac: papules, pustules, erosions (Fig. 26-11). Maculopapular lesions with diffuse erythema. Edema, ulcerations, and fissuring of prepuce, usually in diabetic men; white plaques under foreskin.

Differential Diagnosis

VulvovaginalCandidiasis. Trichomoniasis(caused by *T. vaginalis*), bacterial vaginosis (caused by replacement of normal vaginal flora by an

nus

Diagnosis

Clinical suspicion confirmed by KOH preparation of scraping from mucosal surface.

Treatment

Azole Creams or Suppository. Treat sexual partners and consider systemic therapy (as for mucocutaneous candidiasis p. 596) if recurrent.

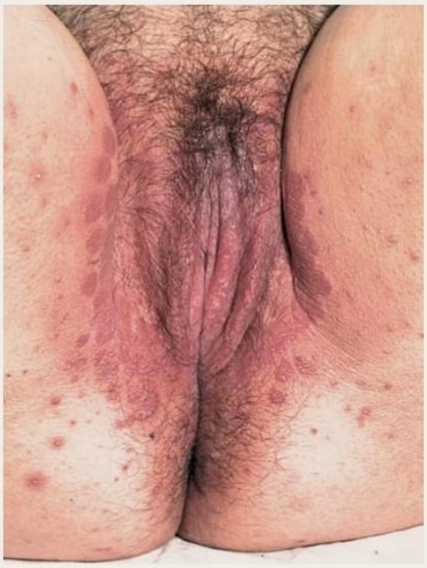

Figure 26-10. Candidiasis: vulvitis and intertrigo Psoriasiform, erythematous lesions becoming confluent on the vulva with erosions and satellite pustules on the thighs.

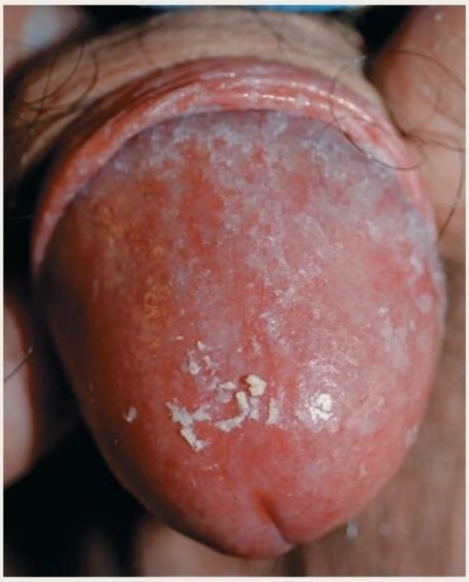

Figure 26-11. Candidiasis: balanoposthitis A 52-year-old uncircumcised male. Erythema and a curd-like matter is seen on the glans penis and foreskin.

Chronic Mucocutaneous Candidiasis
ICD-9: 112.3 ∘ ICD-10: B37.7

- Characterized by persistent or recurrent Candida infections of the oropharynx, skin, and nail apparatus.
- **Inheritance.** Usually autosomal recessive or sporadic.
- **Host Defense Defect.** Various specific and global defects in cell-mediated immunity.
- **Onset.** Usually in infancy or early childhood.

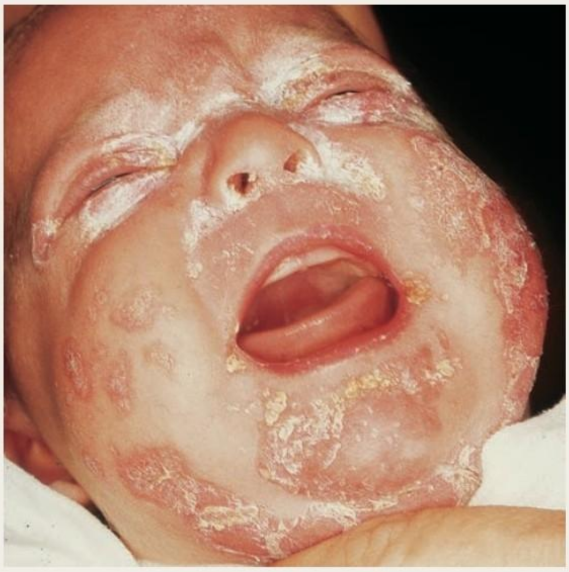

Figure 26-12. Mucocutaneous candidiasis Persistent candidiasis in an immunocompromised infant manifesting as erosions covered by scales and crusts, oropharyngeal candidiasis, and widespread infection of the trunk.

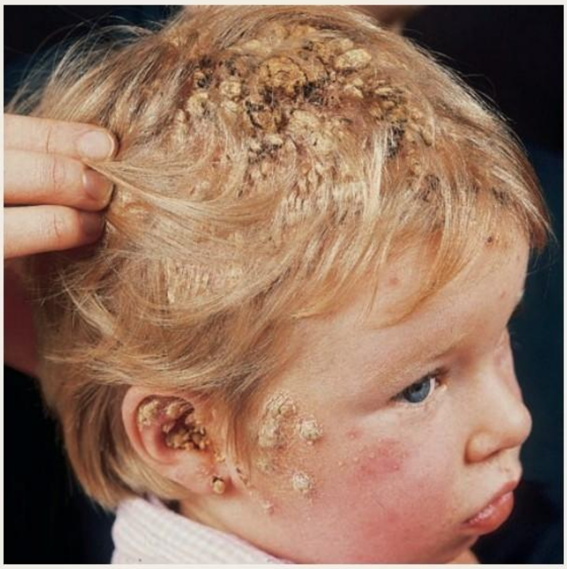

Figure 26-13. Mucocutaneous candidiasis A 3-year-old child with hypothyroidism had thrush, intertriginous candidiasis, warty hyperkeratoses, and crusts on the scalp and face; and also, candidal onychomycosis.

Disseminated Candidiasis ICD-9: 112.5 ◦ ICD-10: B37 ◧ ○

- **Etiology.** *C. albicans, C. tropicalis,* and other non-*albicans* species.
- **Incidence.** Fifth most common cause of nosocomial bloodstream infections in the United States.
- **Risk Factors.** Neutropenia. Venous access
- **Pathogenesis.** *Candida* enters the blood stream having colonized venous access catheters or penetrated the intestinal mucosa. Candidemia seeds the skin and internal organs, i.e., hepatosplenic candidiasis.

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Clinical Manifestation

Cutaneous Lesions. Small disseminated erythematous cutaneous papules (Fig. 26-14). Lesions may occur acutely or chronically.

Systemic Dissemination. Eye with retinal changes. Liver, spleen, CNS

Differential Diagnosis

Malassezia folliculitis, which occurs on the trunk of healthy individuals.

Diagnosis

Lesional biopsy specimen: *Candida* yeast forms are visualized in the dermis; *Candida* species isolated on culture.

Course

Candidemia has high associated morbidity and mortality.

Treatment

Fluconazole in nonneutropenic patients; triazoles echinocandins, caspofungin, micafungin, anidulafungin, voriconazole and posaconazole, as well as lipid formulations of amphotericin B.

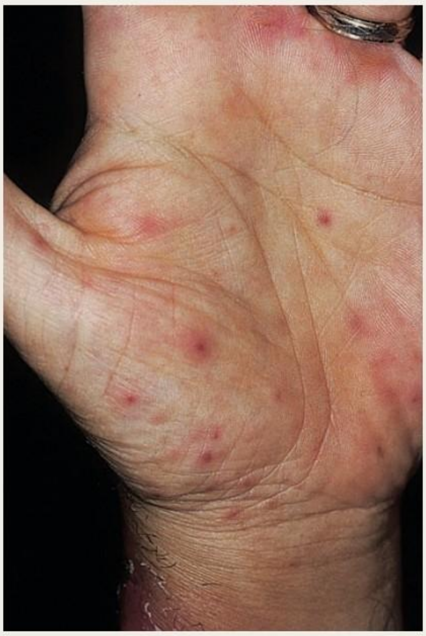


Figure 26-14. Invasive candidiasis with candidemia Multiple, erythematous papules on the hand of a febrile patient with granulocytopenia associated with treatment of acute myelogenous leukemia. The usual source of the infection is the gastrointestinal tract. *C. tropicalis* was isolated on blood culture; candidal forms were seen on lesional skin biopsy.

Tinea Versicolor ICD-9: 111.0 ◦ ICD-10: B36.0 ☐ ●

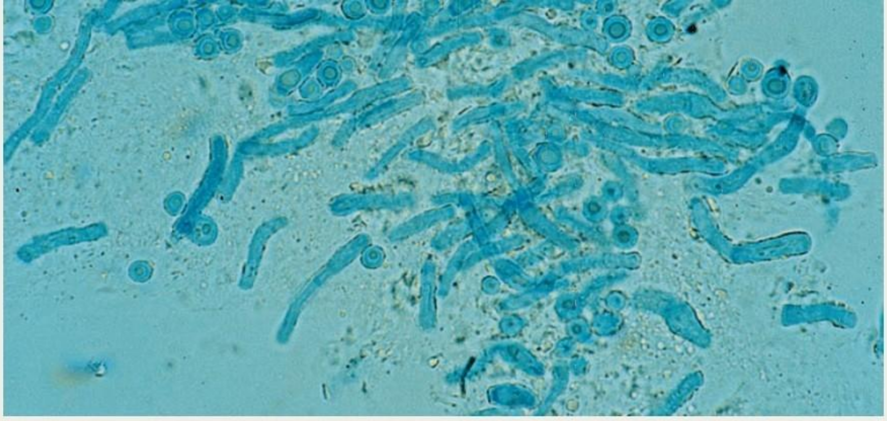


Figure 26-15. ***Malassezia furfur*****: KOH preparation** Round yeast and elongated pseudohyphal forms, so-called "spaghetti and meatballs."

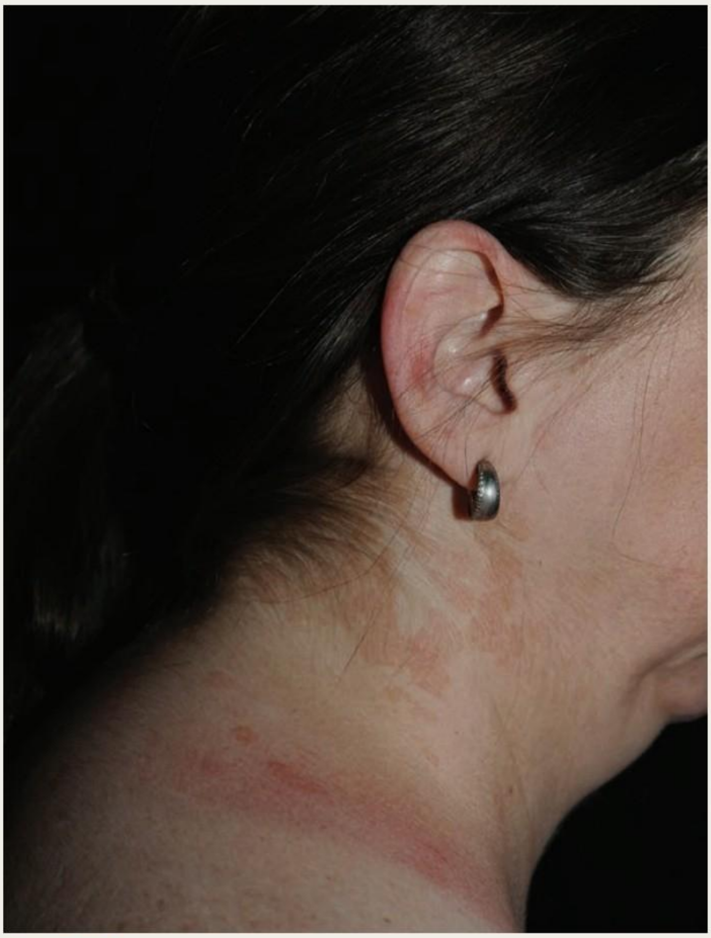


Figure 26-16. **Pityriasis versicolor** A 43-year-old white female with orange-tan lesions of the lateral neck. Sharply marginated scaling macules.

Color. In nontanned skin, lesions are *light brown* (Fig. 26-18) or pink. On tanned skin, *hypopigmented* (Fig. 26-19). In brown- or black-skinned persons, *dark brown macules* (Figs. 26-17 and 26-20). Brown of varying intensities and hues (Fig. 26-18). In time, individual lesions may enlarge and merge, forming extensive *geographic areas*.

Distribution. Upper trunk, upper arms, neck, abdomen, axillae, groins, thighs, genitalia. Facial, neck, or scalp lesions occur in persons applying creams or ointments or topical glucocorticoid preparations.

Differential Diagnosis

Hypopigmented Macules. Vitiligo, pityriasis alba, postinflammatory hypopigmentation.

Scaling Lesions. Tinea corporis, seborrheic dermatitis, cutaneous T cell lymphoma.

Laboratory Examinations

Direct Microscopic Examination of Scales Prepared with KOH. Filamentous hyphae and globose yeast forms, termed *spaghetti and meatballs* are seen (Fig. 26-15).

cans associated with obesity.

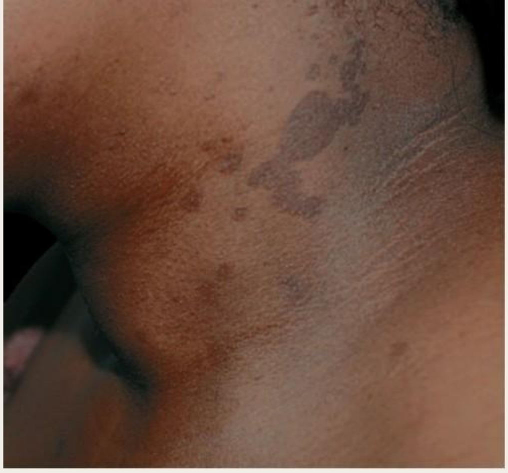
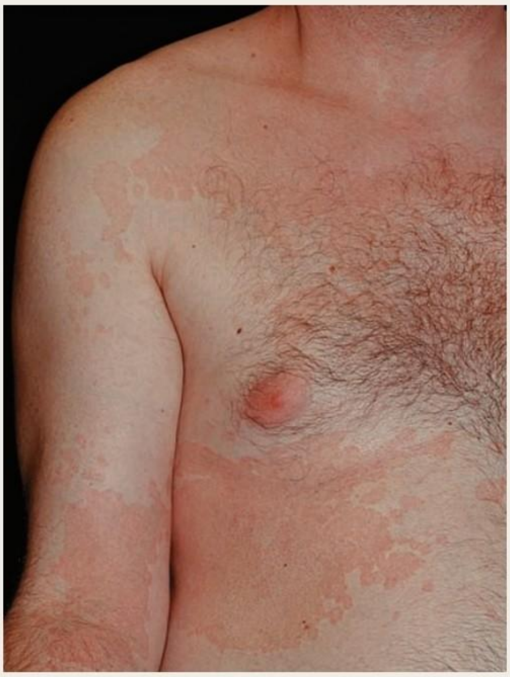


Figure 26-18. Pityriasis versicolor: chest and arm A 36-year-old male with pigmented patches on chest and arms for several years. Multiple pink, well-demarcated scaling macules becoming confluent on the neck, chest, flank, and arm.



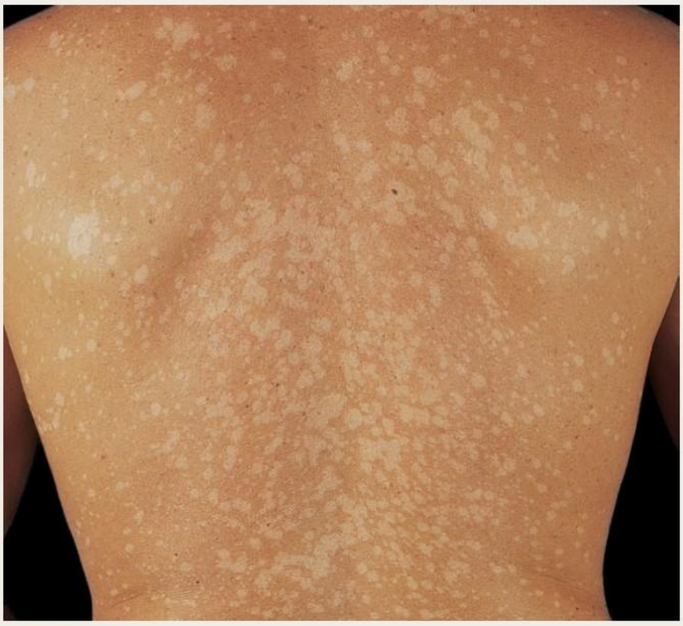


Figure 26-19. Pityriasis versicolor: back Multiple, small-to-medium-sized, well-demarcated hypopigmented macules on the back of a tanned individual with white skin.

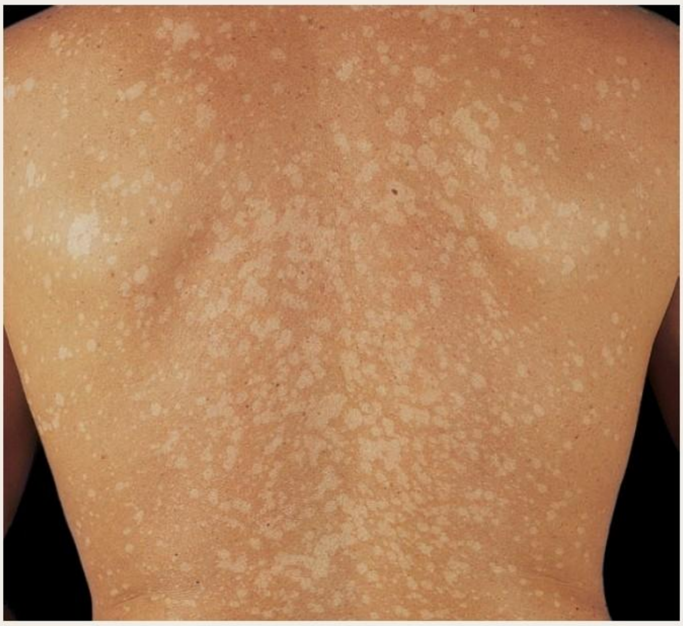


Figure 26-19. Pityriasis versicolor: back Multiple, small-to-medium-sized, well-demarcated hypopigmented macules on the back of a tanned individual with white skin.

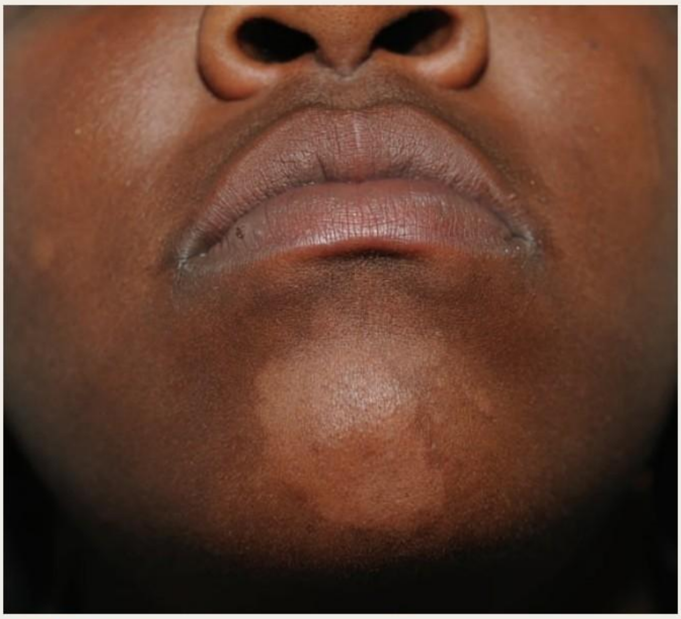


Figure 26-20. Pityriasis versicolor: face A 18-year-old black female hypopigmented scaling macule on chin. She had been applying cocoa butter to face since childhood.

Wood's Lamp. Blue-green fluorescence of scales; may be negative in individuals who have showered recently because the fluorescent chemical is water soluble. Vitiligo appears as depigmented, white, and has no scale.

Dermatopathology. Budding yeast and hyphal forms in the most superficial layers of the stra-

for months after infection has been eradicated.

Treatment

Topitcal agents. Selenium sulfide (2.5%) lotion

5–20% *solution* is applied at the edge of coverslip. Capillary action draws solution under

gastric pH for dissolution of tablet. Take with food or cola beverage; antacids and H_2

blockers reduce absorption. The most hepatotoxic of azole drugs; hepatotoxicity occurs in an estimated one of every 10,000–15,000 exposed persons. Not approved for treatment of dermatophyte infections in the United States.

Dermatophytoses of Epidermis

Epidermal dermatophytoses are the most common dermatophytic infection. May be associated with dermatophytic infection of hair/hair follicles and/or the nail apparatus. *Synonym*: Ringworm.

Tinea Pedis ICD-9: 110.4 ∘ ICD-10: B35.3 ☐ ◐

- Dermatophytic infection of the feet.
- **Clinical Findings.** Erythema, scaling, maceration, and/or bulla formation. Infections at other sites such as tinea cruris usually associate initial tinea pedis.
- **Course.** Provides breaks in the integrity of the epidermis through which bacteria such as *S. aureus* or group A streptococcus (GAS) can invade, causing skin or soft-tissue infection.
- **Synonyms.** Athlete's foot. Jungle rot.

Epidemiology

Age of Onset. Late childhood or young adult life. Most common, 20–50 years.

Predisposing Factors. Hot, humid climate; occlusive footwear; hyperhidrosis.

Clinical Manifestation

Duration: months to years to lifetime. Often, prior history of tinea pedis, and tinea unguium of toenails. Usually asymptomatic. Pruritus. Pain with secondary bacterial infection (Fig. 25-30).

Interdigital Type. Two patterns: dry scaling (Fig. 26-26); maceration, scaling, fissuring of toe webs (Fig. 26-27). Hyperhidrosis common. Most common site: between fourth and fifth toes. Infection may spread to adjacent areas of feet.

Moccasin Type. Well-demarcated scaling with erythema with minute papules on margin, fine white scaling, and hyperkeratosis (Figs. 26-28 and 26-29) (confined to heels, soles, lateral

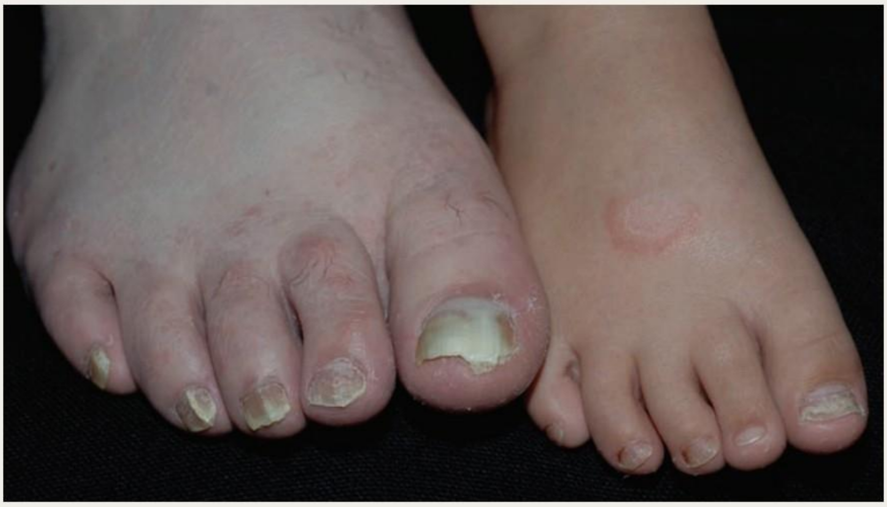


Figure 26-25. Tinea pedis and onychomycosis in father and son The foot of a 5-year-old male with tinea pedis (ringworm lesion) and toenail dystrophy shown with his father's foot with similar, but more advanced, findings. The son most likely became infected with dermatophyte from fomite in his home. Both father and son had atopic diathesis with history of atopic dermatitis.

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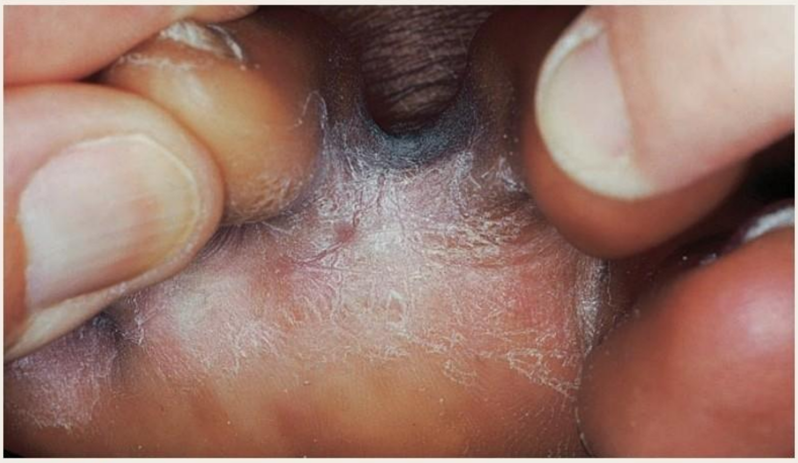


Figure 26-26. Tinea pedis: interdigital dry type The interdigital space between the toes shows erythema and scaling; the toenail is thickened, indicative of associated distal subungual onychomycosis.

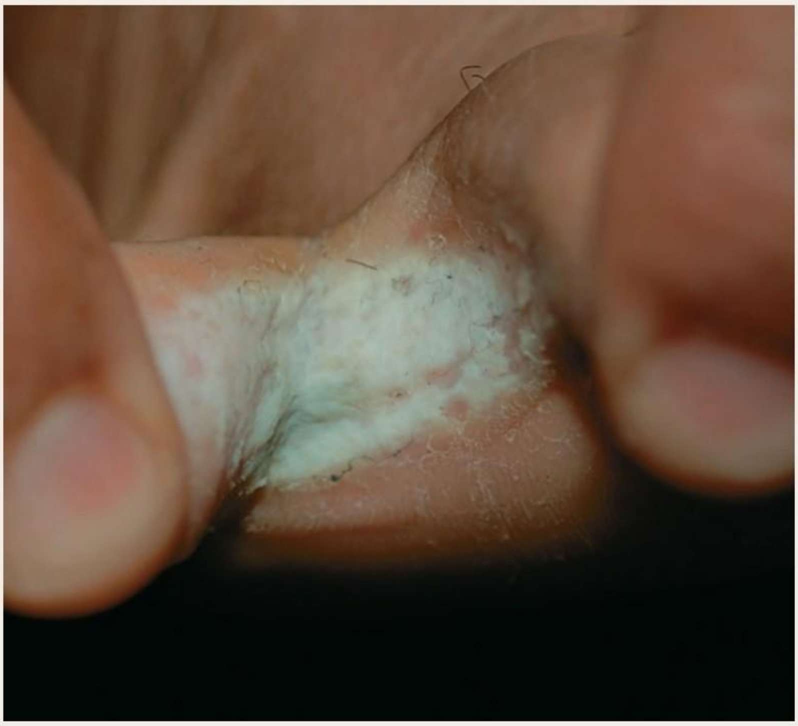
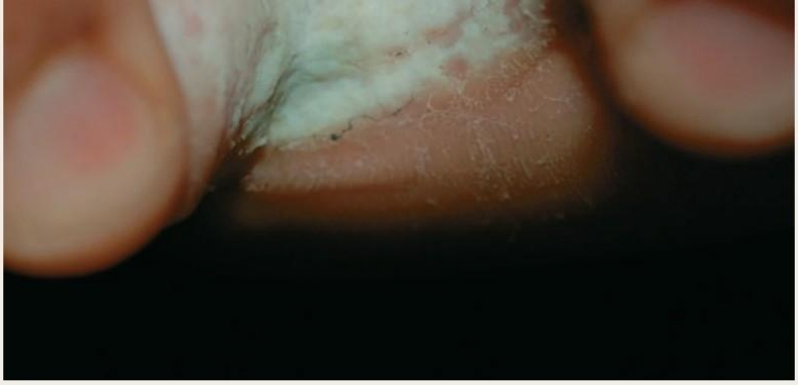


Figure 26-27. Tinea pedis: interdigital macerated type A 48-year-old male with athlete's foot and hyperhidrosis for years. The skin of the webspace between the fourth and fifth toes is hyperkeratotic and macerated (hydration of the stratum corneum). The KOH+ preparation shows septated hyphae, confirming the diagnosis of dermatophytosis. Wood's lamp demonstrated coral-red fluorescence confirming concomitant erythrasma. *P. aeruginosa* was isolated on bacterial culture.



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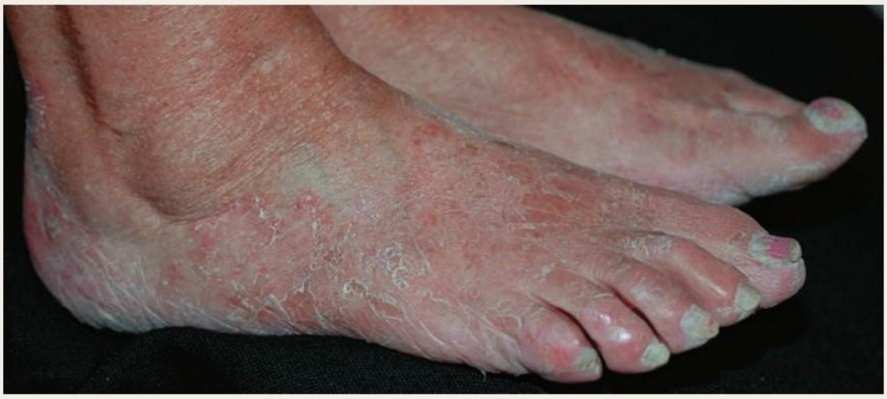

Figure 26-28. Tinea pedis: moccasin type A 65-year-old female with scaling feet for years. Sharply marginated erythema of the foot with a mild keratoderma associated with distal/lateral subungual onychomycosis, typical of *T. rubrum* infection.

borders of feet). *Distribution*: Sole, involving area covered by a *ballet slipper*. One or both feet may be involved with any pattern; bilateral involvement more common.

Inflammatory/Bullous Type. Vesicles or bullae filled with clear fluid (Fig. 26-30). Pus usually indicates secondary infection with *S. aureus* infection or GAS. After rupturing, erosions with ragged ringlike border. May be associated with "id" reaction (autosensitization or dermatophytid). *Distribution*: Sole, instep, webspaces.

Ulcerative Type. Extension of interdigital tinea pedis onto plantar and lateral foot (Fig. 26-30). May be secondarily by *S. aureus*.

Differential Diagnosis

Interdigital Type. Erythrasma, pitted keratolysis

Moccasin Type. Psoriasis, eczematous dermatitis (dyshidrotic, atopic, allergic contact), pitted keratolysis.

Inflammatory/bullous type. Bullous impetigo, allergic contact dermatitis, dyshidrotic eczema, bullous disease.

Laboratory Examinations

Direct Microscopy (Fig. 26-24). In bullous type, examine scraping from the inner aspect of bulla roof for detection of hyphae.

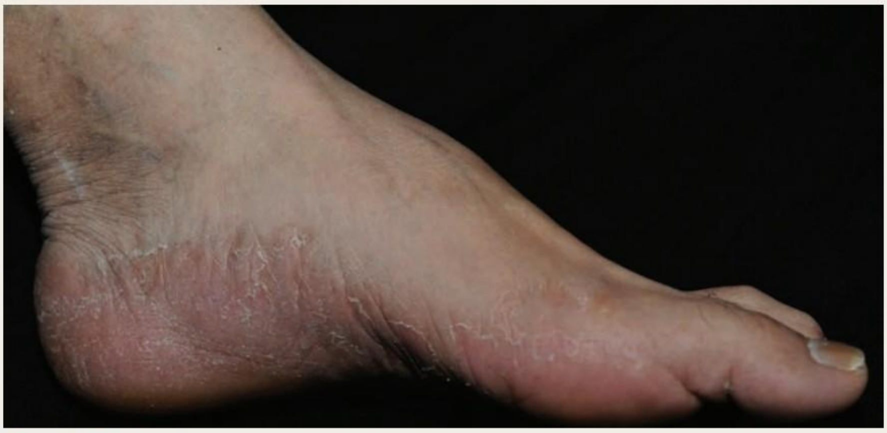

Figure 26-29. Tinea pedis: moccasin type A 63-year-old male with scaling feet for years. Sharply marginated erythema of the medial foot with a mild keratoderma. Tinea corporis was also present on the forearms and dorsum of hands.

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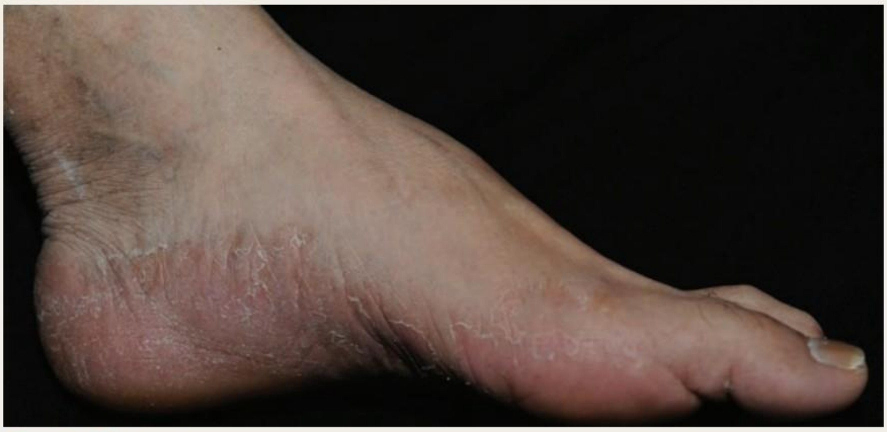

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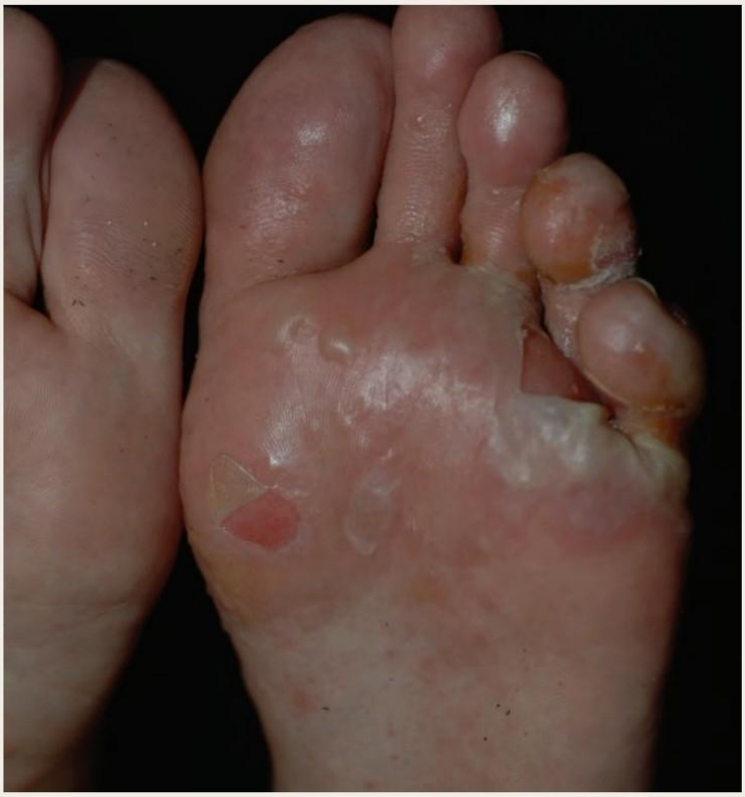


Figure 26-30. Tinea pedis: bullous and ulcerative types A 34-year-old female with painful blisters in the webspaces and on the plantar foot. Tinea pedis was secondarily infected with *S. aureus*. A dermatophytid reaction was present on the hands with small vesicle on the fingers.

Wood's Lamp. Negative fluorescence usually rules out erythrasma in interdigital infection. Erythrasma and interdigital tinea pedis may coexist.

Culture. Dermatophytes can be isolated in 11% of normal-appearing interspaces and 31%

Course

Tends to be chronic. May provide portal of entry for soft-tissue infections, especially in patient's venous stasis. Without secondary prophylaxis, recurrence is the rule.

Borders well demarcated; central clearing. May extend onto dorsum of hand with follicular papules, nodules, and pustules with dermatophytic folliculitis. *Dyshidrotic type*: Papules, vesicles, bullae (uncommon on the margin of lesion) on

ment of palmar creases or patchy scaling on the dorsa and sides of fingers; 50% of patients have unilateral involvement. Usually associated with tinea pedis (Fig. 26-32) and tinea cruris. If chronic, often associated with tinea unguium of fingernails and toenails (Fig. 26-32).

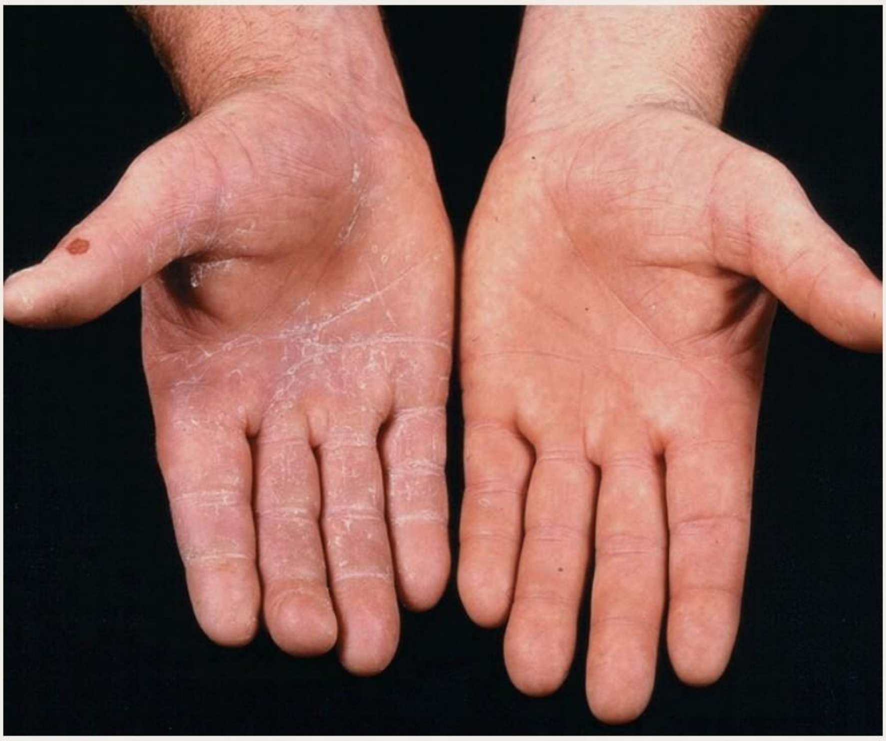


Figure 26-31. Tinea manuum Erythema and scaling of the right hand, which was associated with bilateral tinea pedis; the "one-hand, two-feet" distribution is typical of epidermal dermatophytosis of the hands and feet. In time, distal/lateral subungual onychomycosis occurs on the fingernails.

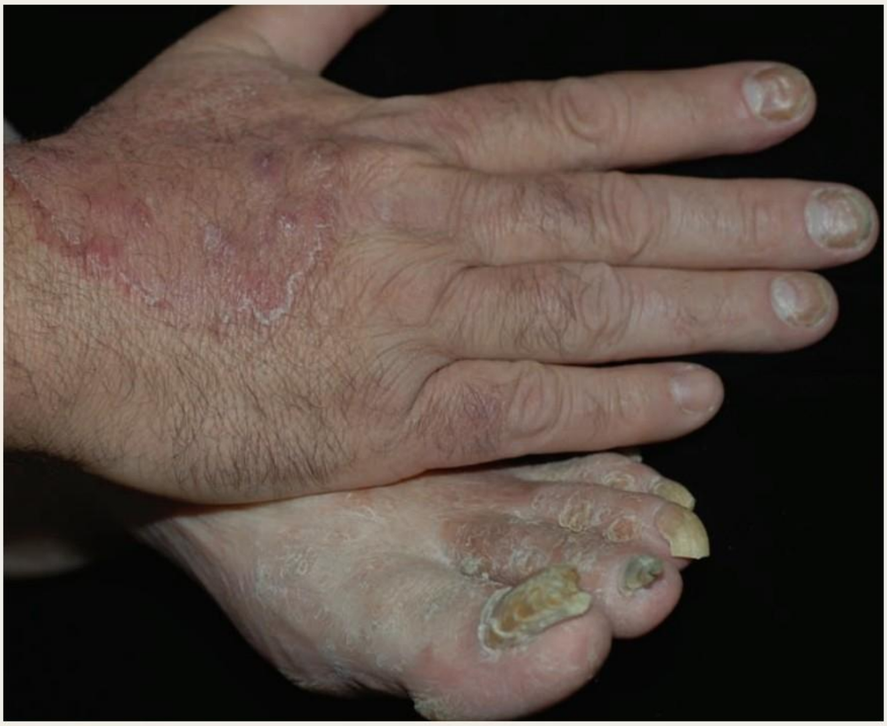


Figure 26-32. Tinea manuum, tinea pedis, and onychomycosis A 57-year-old male immunosuppressed renal transplant recipient with extensive epidermal dermatophytosis of hands, feet, and nail. The feet are initially infected; infection spreads to hands, arms, and nails.

Differential Diagnosis

Atopic dermatitis, lichen simplex chronicus, allergic contact dermatitis, irritant contact der-

Treatment

Must eradicate tinea unguium of fingernails as well as toenails; also tinea pedis and tinea cruin darker-skinned persons. In atopics, chronic scratching may produce secondary changes of lichen simplex chronicus. *Distribution.* Groins and thighs; may extend to buttocks (Figs.

Prevention. After eradication minimize reinfection with shower shoes and antifungal powders; **Antifungal Agents.** See p. 609

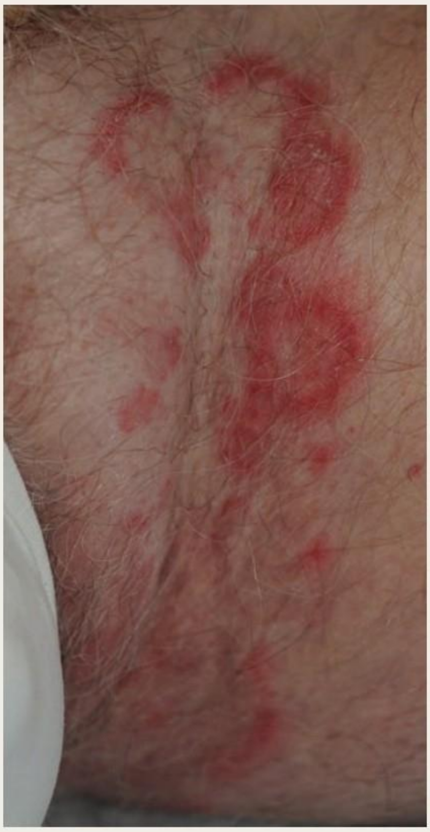


Figure 26-33. Tinea cruris (inguinalis): acute A 80-year-old female with pruritic inguinal rash for several weeks. She was being treated with prednisone for polymyalgia rheumatica. Typical inflamed rings and arcs are seen on the proximal thigh and adjacent inguinal area.

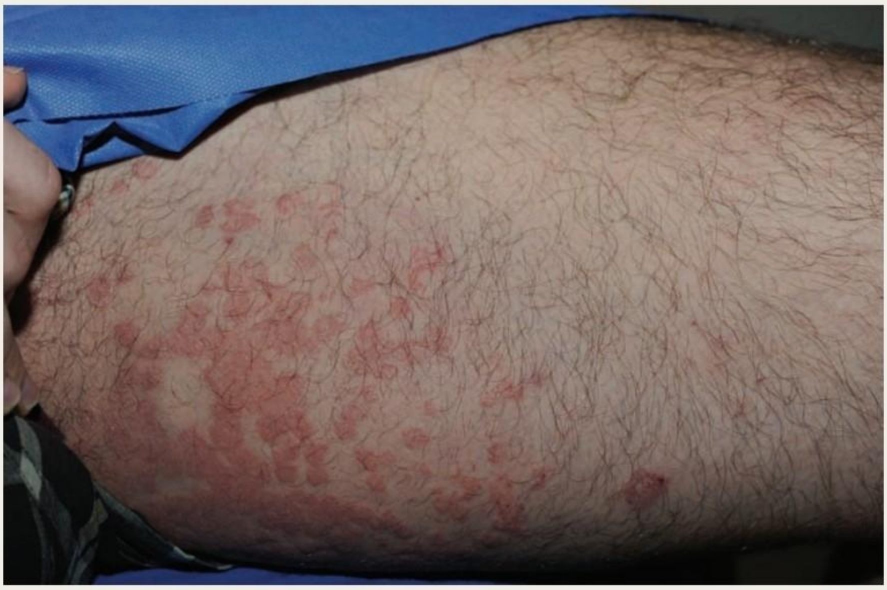


Figure 26-34. Tinea cruris (inguinalis): subacute A 20-year-old male with pruritic inguinal rash for several months. He was a college wrestler. Concomitant dermatophyte infection was also present on the feet, trunk, and face. He was treated with oral terbinafine.

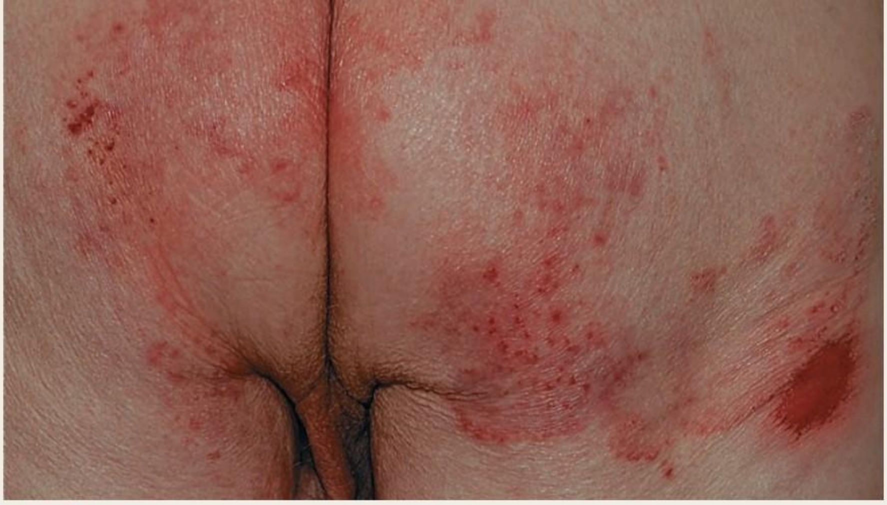
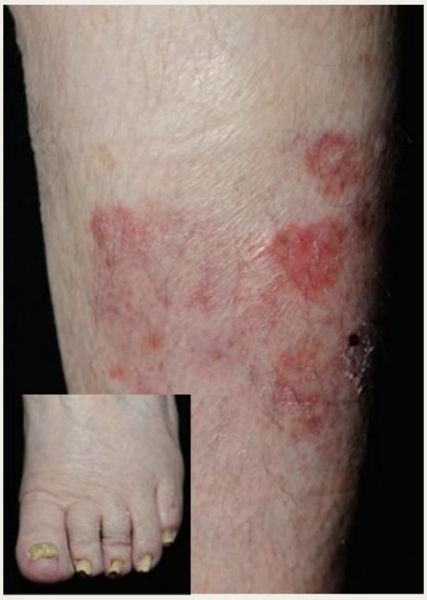


Figure 26-36. Tinea corporis: tinea incognito An 80-year-old male with a rash on buttocks for 1 year. Erythematous patches on the buttocks, some with sharp margination, others with clearing, and excoriations. He had been treating the pruritus with topical corticosteroid. Tinea cruris, tinea pedis, and onychomycosis were also present.

Figure 26-37. Tinea corporis A 80-year-old female with red, scaling lesions on the lower leg. Lesions were present under a foot brace that occluded the skin. Corticosteroid has been applied to the site. Tinea corporis was associated with tinea pedis and onychomycosis (see inset).



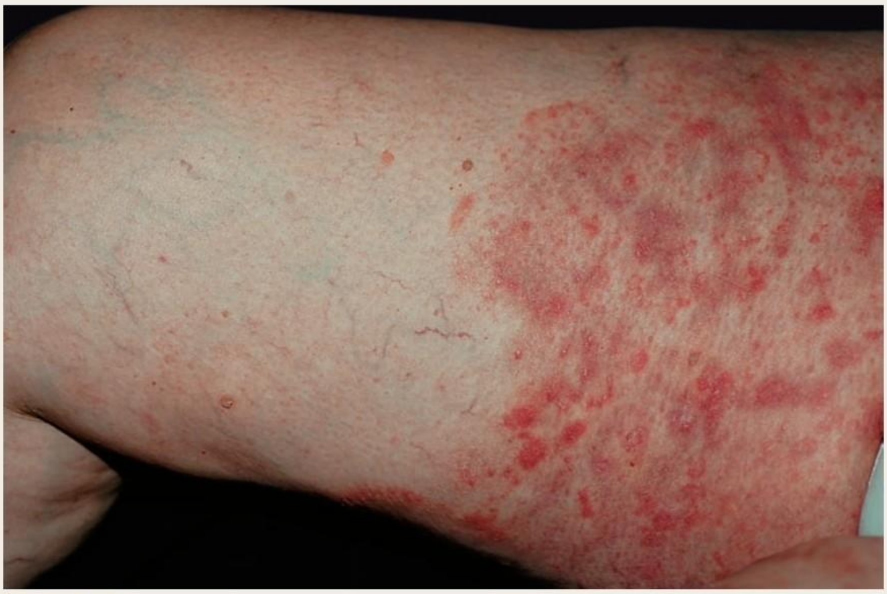
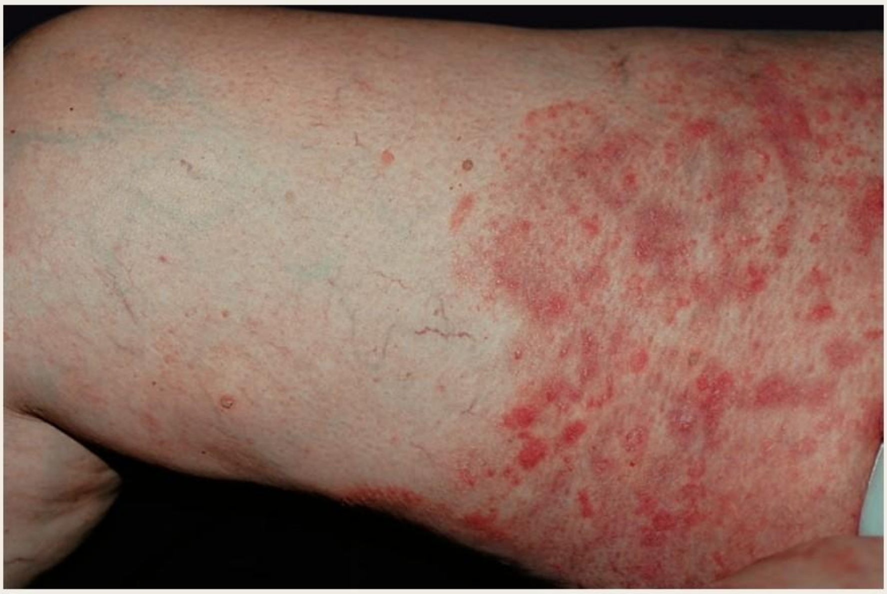


Figure 26-38. Tinea corporis: tinea incognito A 60-year-old renal transplant recipient has been treating thigh rash with topical corticosteroid for several months. Blotchy erythema with areas of atrophy and scale on the right medial upper thigh bordering the inguinal area. Tinea pedis and onychomycosis were also present. KOH preparation showed septated hyphae. Topical steroid facilitates dermatophyte growth, suppressing the immune response, creating an undiagnosed infection, tinea incognito.



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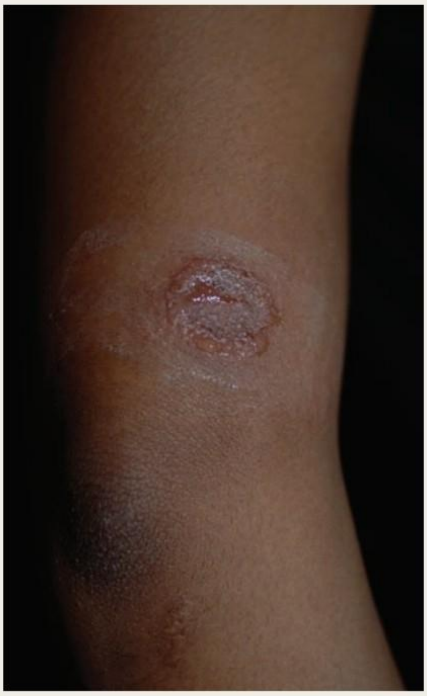


Figure 26-39. Tinea corporis: inflammatory A 13-year-old female with inflammatory lesion on the arm for 1 week. A younger sibling had tinea capitis. Acutely inflamed edematous exudative annular plaque on the upper arm.

Tinea Facialis ◧ ◐

- Dermatophytosis of the glabrous facial skin. Well-circumscribed erythematous patch. More commonly misdiagnosed than any other dermatophytosis.
- *Synonym*: Tinea faciei
- **Etiology.** *T. tonsurans* associated with tinea capitis in black children and their parents. *T. mentagrophytes, T. rubrum* most commonly; also *M. audouinii, M. canis.*

Clinical Manifestation

Well-circumscribed macule to plaque of variable size; elevated border and central regression (Figs. 26-40 and 26-41). Scaling is often minimal. Pink to red; in black patients, hyperpigmentation. Any area of face but usually not symmetric.

morphous light eruption, phototoxic drug eruption, lymphocytic infiltrate.

Diagnosis

See “Direct Microscopy,” and culture.

Treatment

thema migrans, lupus erythematosus, poly-

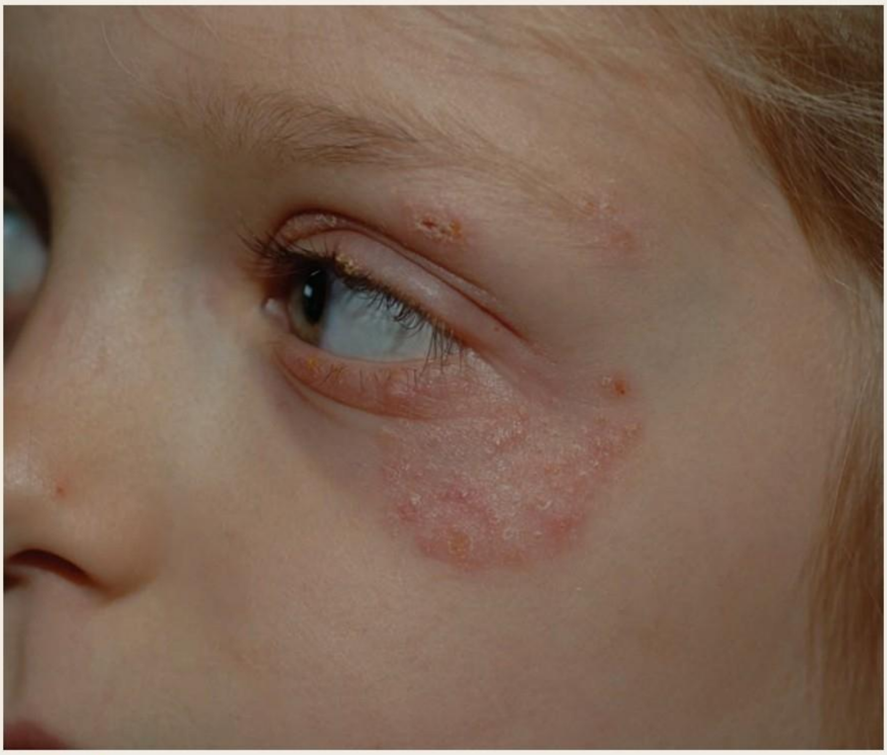


Figure 26-40. Tinea facialis A 5-year-old girl with inflammatory lesion on the periorbital skin. Papules are dermatophytic folliculitis of vellus hairs. The site has previously been treated with hydrocortisone cream.

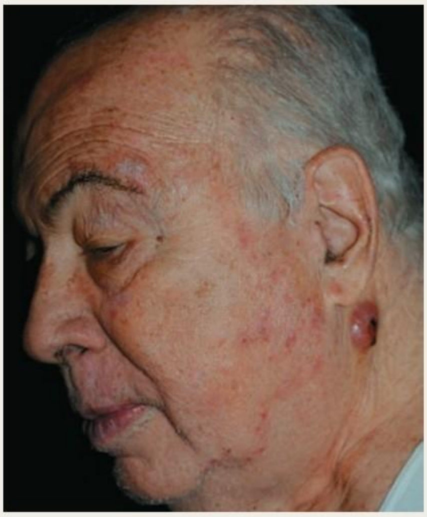


Figure 26-41. Tinea facialis A 83-year-old immunosuppressed male with a history of prednisone treatment for polymyalgia rheumatica and chronic lymphatic leukemia. Note a facial lesions and a new nodule. Well-demarcated erythema and scaling in the beard area. SCC in situ is present on the left eyebrow. The tumor on the left neck is B-cell lymphoma; this lesion regressed when prednisone was tapered.

Tinea Incognito ◧ ◐

- Epidermal dermatophytosis, often associated with dermatophytic folliculitis.
- Occurs after the topical application of a glucocorticoid preparation to a site colonized or infected by dermatophyte.

- Ectothrix infection. Occurs outside hair shaft. Hyphae fragment into arthroconidia, leading to cuticle destruction. Caused by *Microsporum* spp. (*M. audouinii* and *M. canis*) (Fig. 26-42).
- Endothrix infection. Occurs within hair shaft without cuticle destruction (Fig. 26-42). Arthroconidia found within hair shaft. Caused by *Trichophyton* spp. (*T. tonsurans* in North America; *T. violaceum* in Europe, Asia, parts of Africa).
- *"Black dot" tinea capitis*. Variant of endothrix resembling seborrheic dermatitis.

scalp. Tends to be diffuse and poorly circumscribed. Low-grade folliculitis may be present. Resembles seborrheic dermatitis. Usually caused by *T. tonsurans, T. violaceum*. *Differential diagnosis*: Seborrheic dermatitis, psoriasis, atopic dermatitis, lichen simplex chronicus, chronic cutaneous lupus erythematosus, alopecia areata.

Kerion. Inflammatory mass in which remaining hairs are loose. Characterized by boggy, purulent, inflamed nodules, and plaques (Fig. 26-45). Usually painful; drains pus from multiple openings, like honeycomb. Hairs do not break off but fall out and can be pulled without

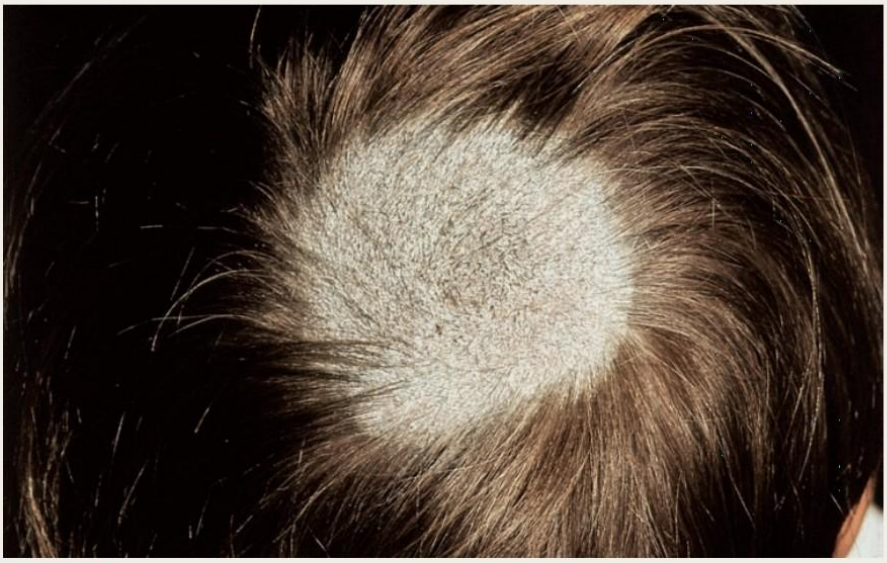


Figure 26-43. Tinea capitis: "gray patch" type A large, round, hyperkeratotic plaque of alopecia due to breaking off of hair shafts close to the surface, giving the appearance of a mowed wheat field on the scalp of a child. Remaining hair shafts and scales exhibit a green fluorescence when examined with Wood's lamp. *M. canis* was isolated on culture.

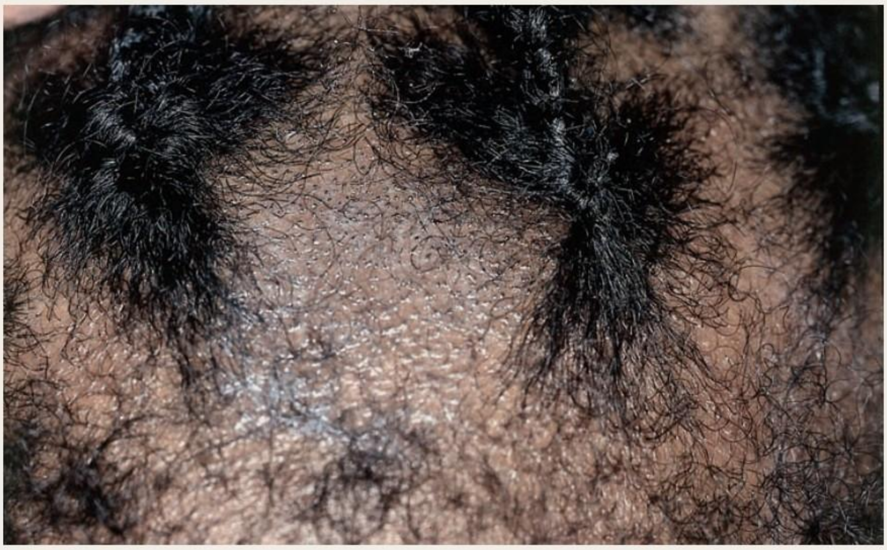


Figure 26-44. Tinea capitis: "black dot" variant A subtle, asymptomatic patch of alopecia due to breaking off of hairs on the frontal scalp in a 4-year-old black child. The lesion was detected because her infant sister presented with tinea corporis. *T. tonsurans* was isolated on culture.

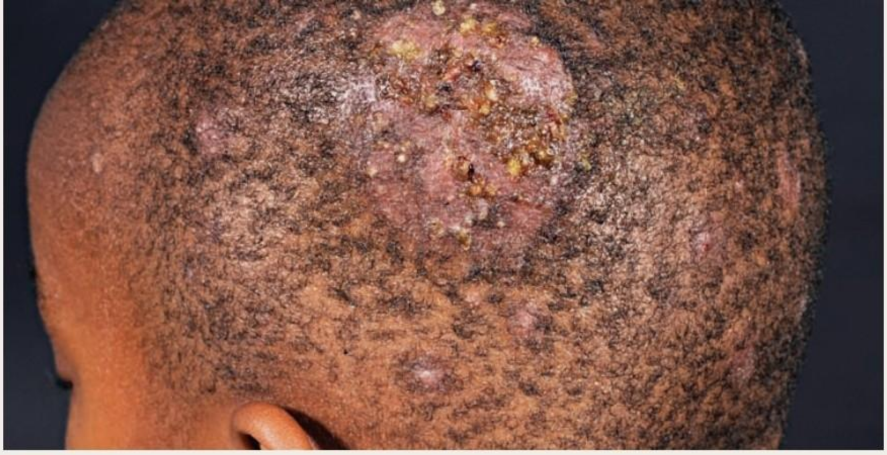


Figure 26-45. Kerion A 5-year-old black boy with an inflammatory mass on the scalp unresponsive to oral antibiotics. The bobby swelling with multiple pustules and postauricular lymphadenopathy. *T. tonsurans* was isolated on fungal culture. He was successfully treated with oral terbinafine for 4 weeks. (From Proudfoot LE, Morris-Jones R. Kerion celsi. *N Engl J Med* 2012;366:1142. Used with permission.)

pain. Follicles may discharge pus; sinus formation; mycetoma-like grains. Thick crusting with matting of adjacent hairs. A single plaque is usual, but multiple lesions may occur with involvement of entire scalp. Frequently, associated lymphadenopathy is present. Usually caused by zoophilic (*T. verrucosum*, *T. mentagrophytes* var. *mentagrophytes*) or geophilic species. Heals with scarring alopecia.

Favus. Latin for honeycomb. Early cases show perifollicular erythema and matting of hair. Later, thick yellow adherent crusts (scutula) composed of skin debris and hyphae that are pierced by remaining hair shafts (Fig. 26-46). Fetid odor. Shows little tendency to clear spontaneously. Often results in scarring alopecia. *Differential diagnosis*: Impetigo, ecthyma, crusted scabies.

Laboratory Examinations

Wood's Lamp. *T. tonsurans* does not fluoresce.

Direct Microscopy. Skin scales contain hyphae and arthrospores. *Ectothrix*: arthrospores can be seen surrounding the hair shaft in cuticle. *Endothrix*: spores within hair shaft. *Favus*: loose chains of arthrospores and airspaces in hair shaft (Fig. 26-42).

Fungal Culture. Growth of dermatophytes usually seen in 10–14 days.

Bacterial Culture. Rule out bacterial infection, usually *S. aureus* or GAS.

Course and Treatment

Chronic untreated kerion and favus, especially if secondarily infected with *S. aureus*, result in scarring alopecia. Regrowth of hair is the rule if treated with systemic antifungal agents (see p. 609).

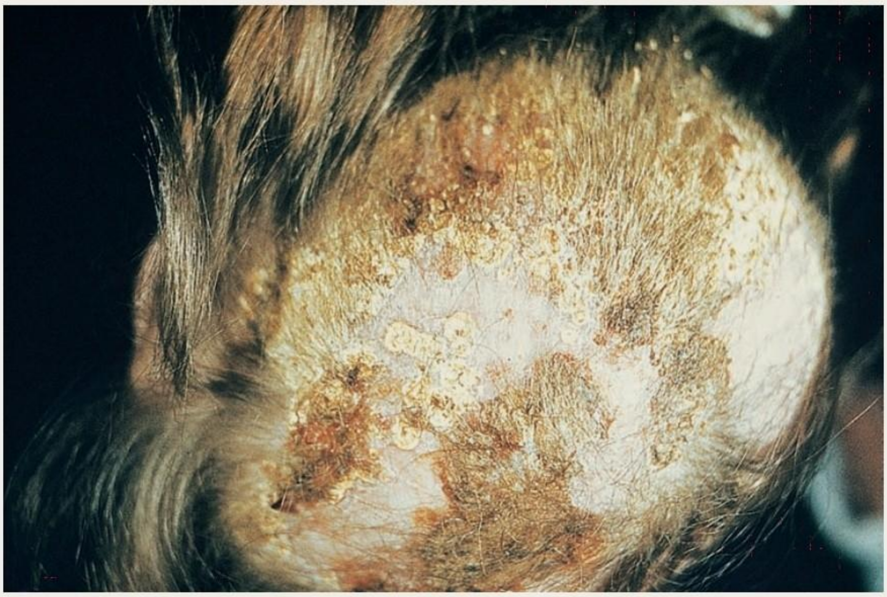


Figure 26-46. Tinea capitis: favus Extensive hair loss with atrophy, scarring, and so-called scutula, i.e., yellowish adherent crusts present on the scalp; remaining hairs pierce the scutula. *T. schoenleinii* was isolated on culture.

pustules, nodules, or plaques. Involved hairs are loose and easily removed. With less follicular involvement, there are scaling, circular, reddish patches (tinea facialis) in which hair is broken off at the surface. Papules may coalesce to inflammatory plaques topped by pustules.

Kerion: boggy purulent nodules and plaques as with tinea capitis (Fig. 26-48).

Treatment

Topical agents ineffective. Systemic antifungal therapy required (see p. 609).

Dermatophytic Folliculitis. See "Infectious Folliculitis" in Section 31.

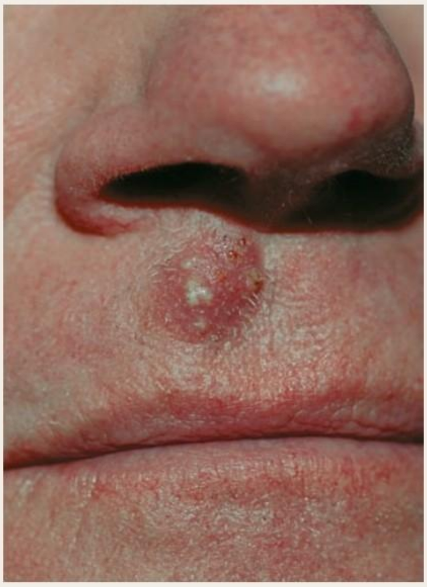


Figure 26-47. Tinea barbae A 63-year-old male with pustules in beard area for several months. A large pustule in an inflammatory nodule is seen on the moustache area. Extensive subtle tinea facialis was also present. Tinea pedis, onychomycosis, and tinea cruris were present as well. KOH preparation was positive; *T. rubrum* was detected on dermatophyte culture. Bacterial culture was negative for pathogens. Facial lesions resolved with oral terbinafine.

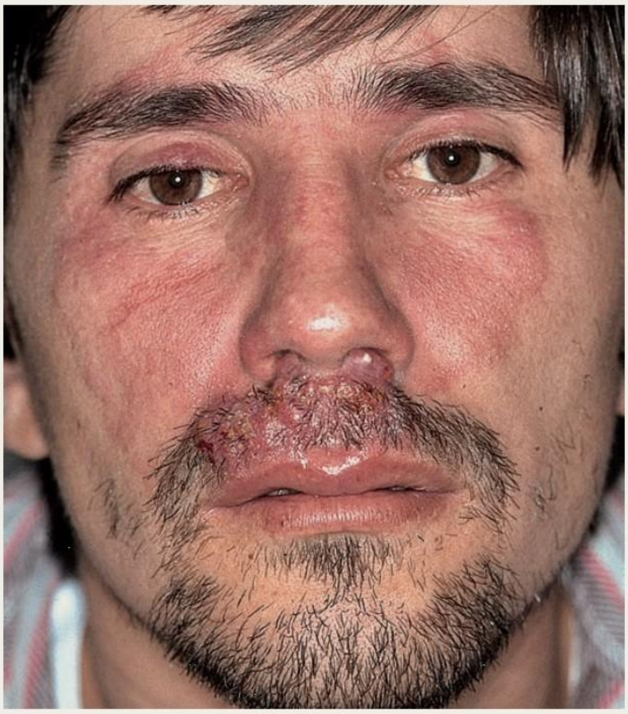


Figure 26-48. Tinea barbae with kerion and tinea facialis Confluent, painful papules, nodules, and pustules on the upper lip (kerion). Epidermal dermatophytosis (tinea facialis) with sharply marginated erythema and scaling is present on the cheeks, eyelids, eyebrows, and forehead. *T. mentagrophytes* was isolated on culture. In this case, the organism caused two distinct clinical patterns (epidermal involvement, tinea facialis versus follicular inflammation, tinea barbae), depending on whether glabrous skin or hairy skin was infected (see also Fig. 26-23).

Majocchi Granuloma ■ ○

- Dermatophytic folliculitis with foreign body

Etiology. Most commonly *T. rubrum*, *T. tonsurans* pox virus. The virus has not been cultured. Not distinguishable from other poxviruses by

Demography. More common in children and sexually active adults; males >females. In advanced human immunodeficiency virus (HIV)

disease, hundreds of small mollusca or giant mollusca occur on the face and other sites.

Pathogenesis. A subclinical carrier state of MCV probably exists in many healthy adults. Unique among poxviruses, MCV infection results in epidermal tumor formation; other human poxviruses cause a necrotic "pox" lesion. Rupture and discharge of the infectious virus-packed cells occur in the umbilication/crater of the lesion.

Clinical Manifestation

Papules, nodules, tumors with central umbilication or depression (Figs. 27-1–27-4). Skin-colored. Round, oval, hemispherical. Isolated single lesion; multiple, scattered discrete lesions; or confluent mosaic plaques. Larger mollusca may have a central keratotic plug, which gives the lesion a central dimple or umbilication. Gentle pressure on a molluscum extrudes the central plug.

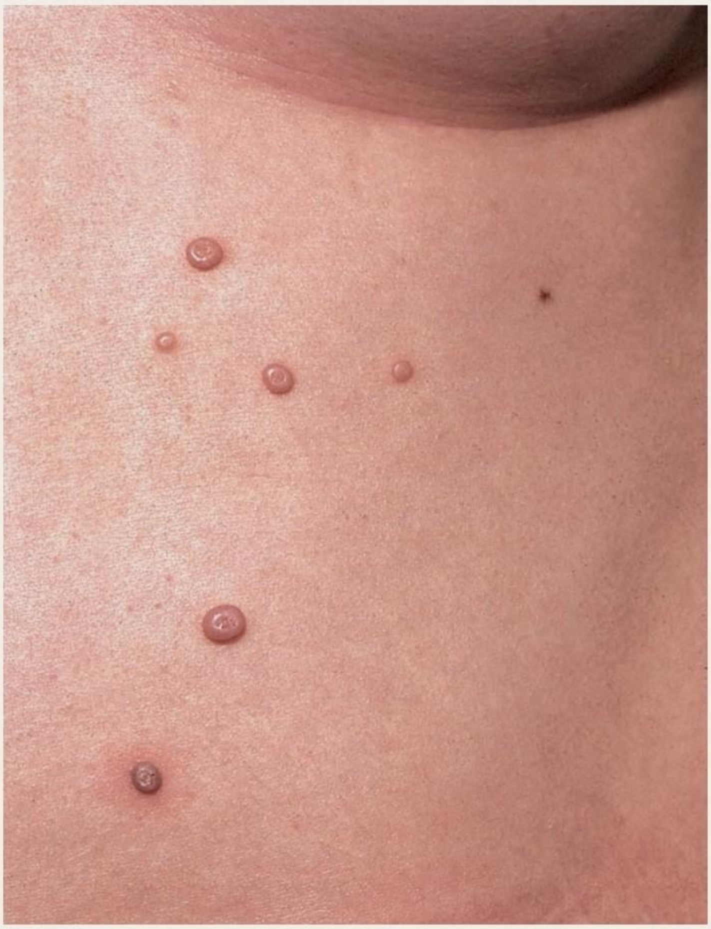
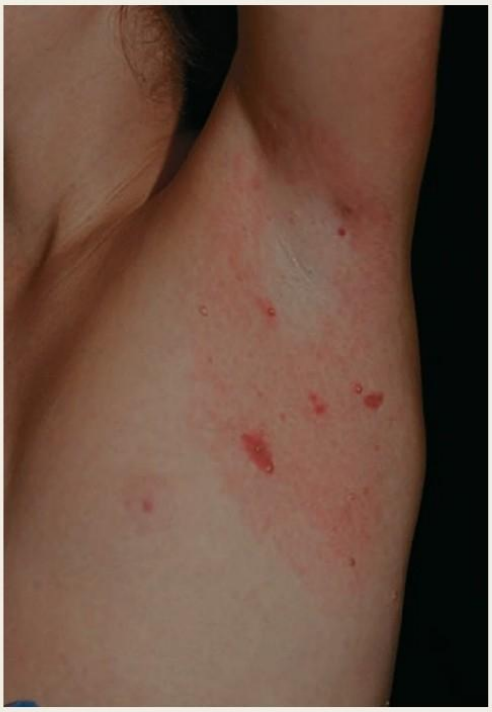


Figure 27-1. Molluscum contagiosum Typical umbilicated papules. Discrete, solid, skin-colored papules 3–5 mm on the chest of an adolescent female. The lesion with red halo is regressing spontaneously.

Figure 27-2. Molluscum contagiosum: axilla Mul-

igure 27-2. **Molluscum contagiosum: axilla** Mul-
ple, small pink papules in the axilla of a healthy child. The
ythema surrounding the lesions represents an inflamma-
ry response to MC and usually indicates the lesions are
gressing.



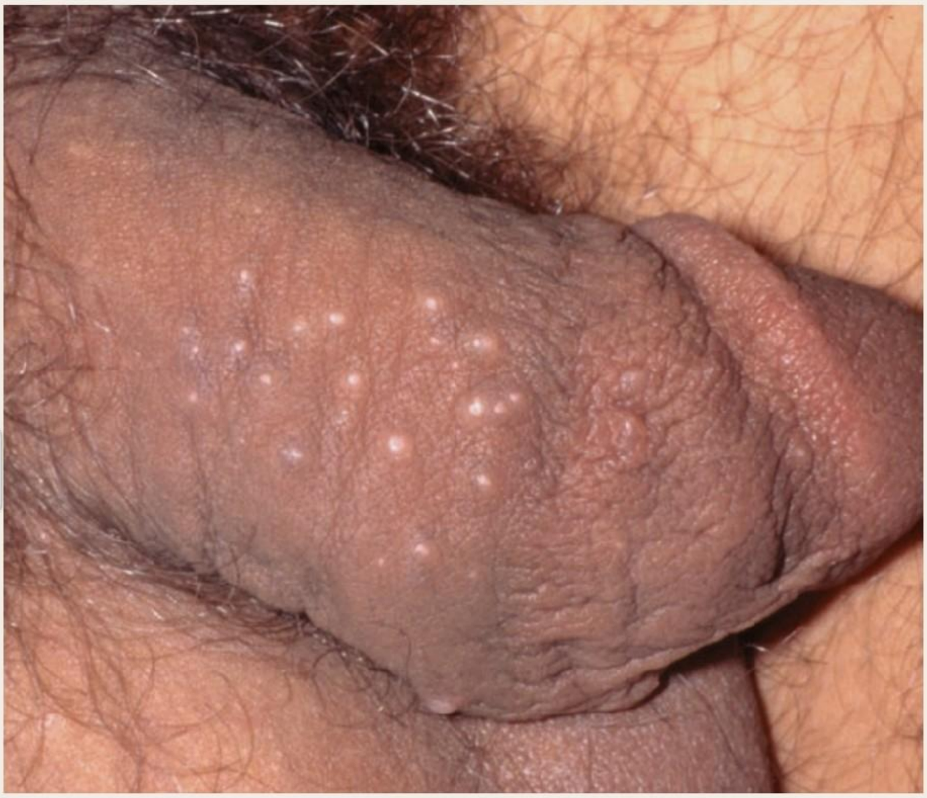


Figure 27-3. **Molluscum contagiosum: penis** Multiple, small shiny papules on penile shaft.

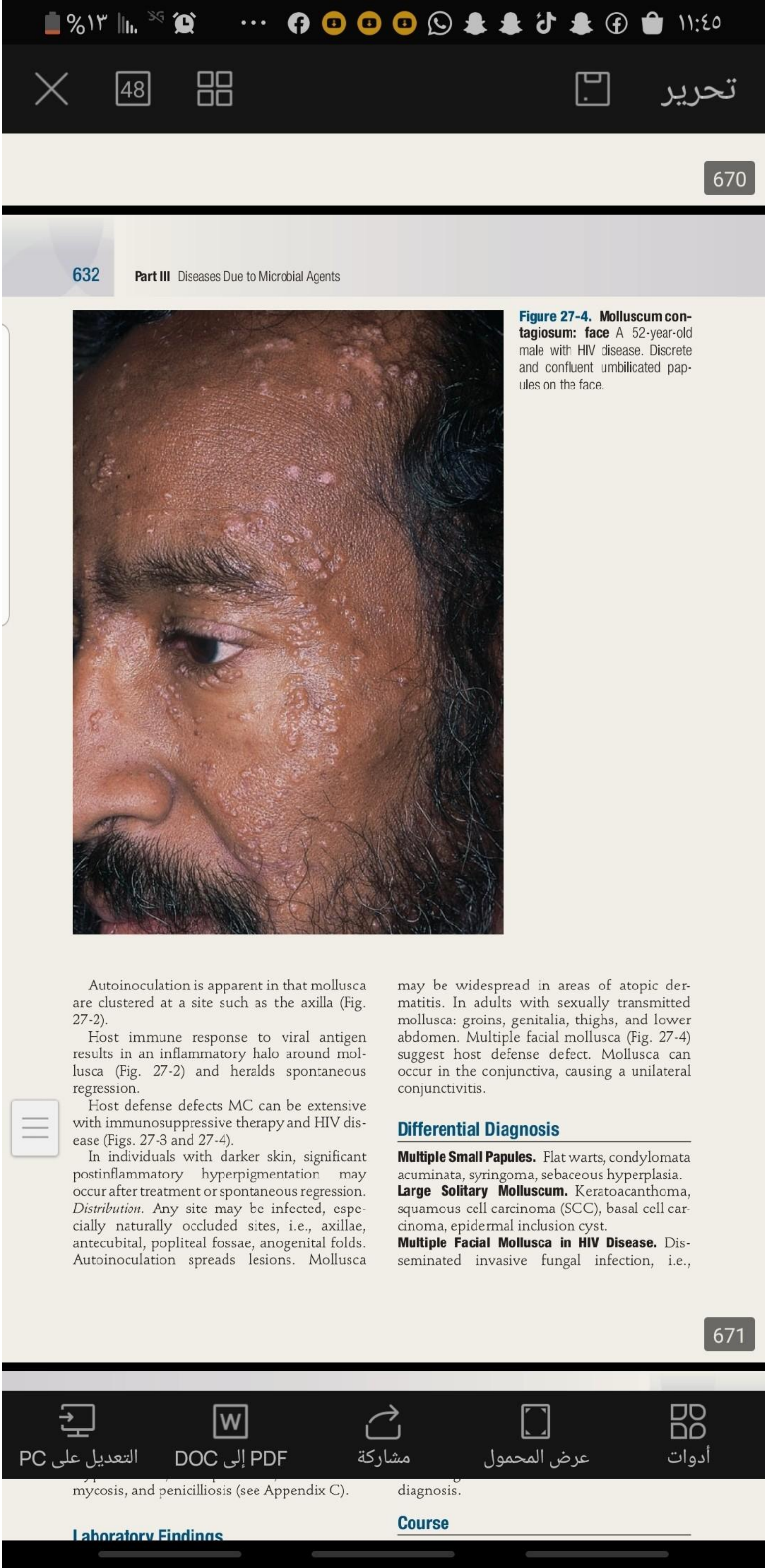


Figure 27-4. Molluscum contagiosum: face A 52-year-old male with HIV disease. Discrete and confluent umbilicated papules on the face.

Autoinoculation is apparent in that mollusca are clustered at a site such as the axilla (Fig. 27-2).

Host immune response to viral antigen results in an inflammatory halo around mollusca (Fig. 27-2) and heralds spontaneous regression.

Host defense defects MC can be extensive with immunosuppressive therapy and HIV disease (Figs. 27-3 and 27-4).

In individuals with darker skin, significant postinflammatory hyperpigmentation may occur after treatment or spontaneous regression. *Distribution.* Any site may be infected, especially naturally occluded sites, i.e., axillae, antecubital, popliteal fossae, anogenital folds. Autoinoculation spreads lesions. Mollusca may be widespread in areas of atopic dermatitis. In adults with sexually transmitted mollusca: groins, genitalia, thighs, and lower abdomen. Multiple facial mollusca (Fig. 27-4) suggest host defense defect. Mollusca can occur in the conjunctiva, causing a unilateral conjunctivitis.

Differential Diagnosis

Multiple Small Papules. Flat warts, condylomata acuminata, syringoma, sebaceous hyperplasia. **Large Solitary Molluscum.** Keratoacanthoma, squamous cell carcinoma (SCC), basal cell carcinoma, epidermal inclusion cyst. **Multiple Facial Mollusca in HIV Disease.** Disseminated invasive fungal infection, i.e.,

mycosis, and penicilliosis (see Appendix C).

Laboratory Findings

diagnosis.

Course

Human Papillomavirus: Cutaneous Diseases □ ● → ◐

- Certain human HPV types commonly infect keratinized skin.
- Cutaneous warts are:
  - Discrete benign epithelial hyperplasia with varying degrees of surface hyperkeratosis.
  - Manifested as minute papules to large plaques.
- Lesions may become confluent, forming a mosaic.
- The extent of lesions is determined by the immune status of the host.

Epidemiology

Transmission. Skin-to-skin contact. Minor trauma with breaks in stratum corneum facilitates epidermal infection.

Demography. Host defense defects are associated with an increased incidence of and more widespread cutaneous warts: HIV disease, iatrogenic immunosuppression with solid organ transplantation.

Epidermodysplasia Verruciformis. Autosomal-recessive hereditary disorder. Acquired EDV-like lesions seen in HIV disease.

Clinical Manifestation

Common Wart or Verruca Vulgaris

Firm papules, 1–10 mm or larger (Figs. 27-11–27-15), hyperkeratotic, clefted surface, with vegetations. Isolated lesion, scattered discrete lesions. Occur at sites of trauma: hands, fingers, and knees. Palmar lesions disrupt the normal line of fingerprints. Return of fingerprints is a sign of resolution of the wart. *Characteristic* "red or brown dots," best visualized with dermatoscope, are pathognomonic, representing thrombosed dermal papilla capillary loops.

Linear arrangement: inoculation by scratching.

Annular warts: at sites of prior therapy.

Butcher's warts: large cauliflower-like lesions on hands of meat handlers.

Filiform warts have relatively small bases, extending out with elongated cap (Fig. 27-11).

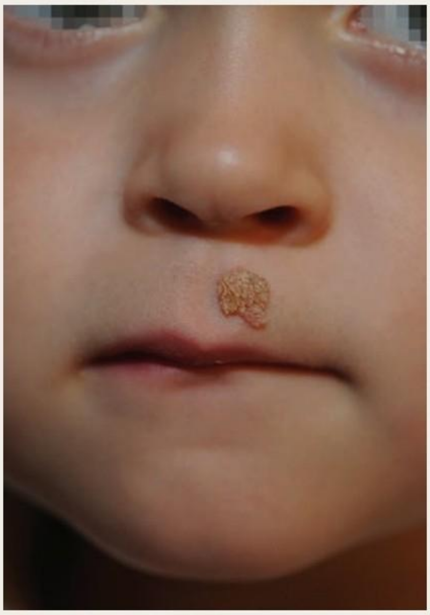


Figure 27-11. Verruca vulgaris on face A 3-year-old boy with common wart on the moustache area.

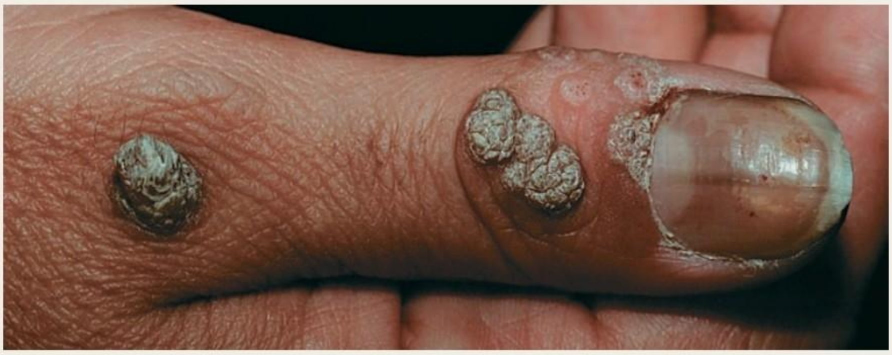
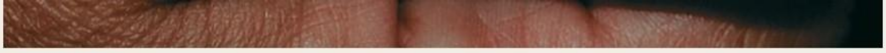


Figure 27-12. Verruca vulgaris: thumb A 25-year-old male with hyperkeratotic, verrucous papules on the dorsal thumb. The dark points represent thrombosed capillaries. The lesion resolved with electrodessication, having failed to respond to cryosurgery.

Plantar Warts (Verruca Plantaris)

Early small, shiny, sharply marginated papule (Fig. 27-16) → plaque with rough hyperkeratotic surface, studded with brown-black dots (thrombosed capillaries). As with palmar warts, normal dermatoglyphics are disrupted. Return of dermatoglyphics is a sign of resolution of the wart. Warts heal without scarring. Therapies such as cryosurgery and electrosurgery can result in scarring at treatment sites. Tenderness may be marked, especially in certain acute types and in lesions over sites of pressure (metatarsal head).

Mosaic warts: Confluence of many small warts. "Kissing" *warts*: lesion may occur on opposing surface of two toes (Fig. 27-17). Plantar foot, often solitary but may be three to six or more. Pressure points, heads of metatarsal, heels, toes.



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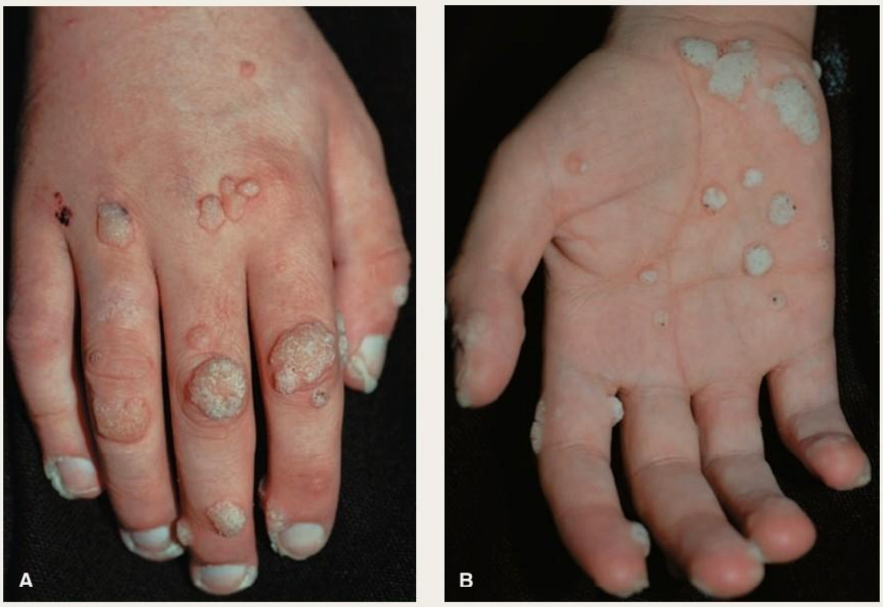

Figure 27-13. Verruca vulgaris: hands A 20-year-old immunosuppressed male with nephrotic syndrome. Multiple verrucae on the **(A)** dorsum and **(B)** palm of the hand.

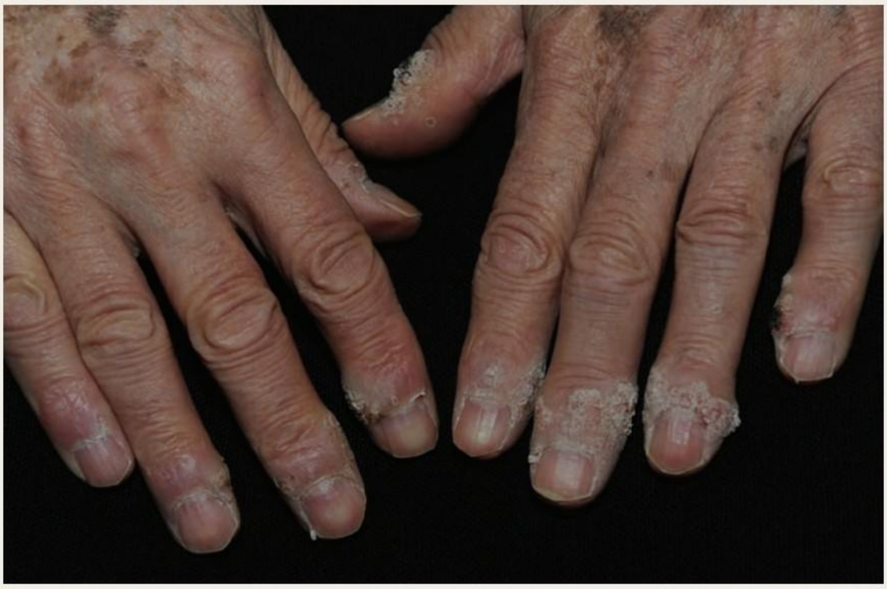

Figure 27-14. Periungual warts A 77-year-old male with extensive periungual warts. He was depressed and picked at periungual skin folds created portal of entry for HPV. Lesions resolved with hyperthermia.

Flat Warts (Verruca Plana)

Sharply defined, flat papules (1–5 mm); "flat" surface; the thickness of the lesion is 1–2 mm (Fig. 27-18). Skin-colored or light brown. Round, oval, polygonal, linear lesions (inoculation of virus by scratching). Occur on face,

and may progress to in situ and invasive SCC.

Human Papillomavirus: Oropharyngeal Diseases

HPV infects mucosal epithelial cells of the hyperkeratosis. Characteristic feature is foci of vacuolated cells (koilocytosis), vertical tiers of parakeratotic cells, and foci of clumped keratohyaline granules.

Diagnosis. Usually made on clinical findings. With host defense defects, HPV-induced SCC at periungual sites or anogenital region should be ruled out by lesional biopsy.

Course

In immunocompetent individuals, cutaneous HPV infections usually resolve spontaneously, without therapeutic intervention. With host defense defects, cutaneous HPV infections may be very resistant to all modalities of therapy. With EDV, lesions first occur at 5–7 years of age and increase in numbers progressively, becoming widespread in some. About 30–50% of individuals with EDV develop malignant cutaneous lesions on areas of skin exposed to sunlight.

Treatment

Goal. Aggressive therapies, which are often quite painful and may be followed by scarring,

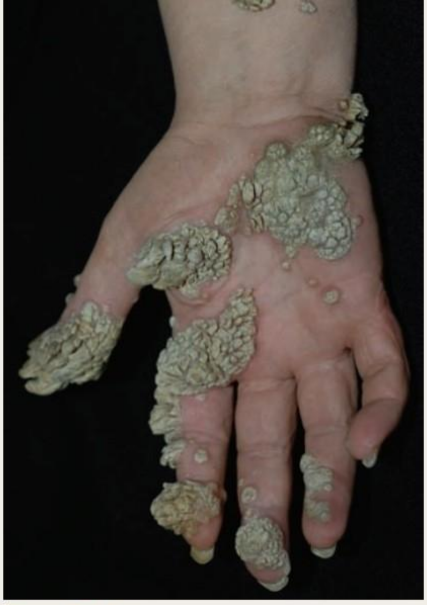

Figure 27-15. Giant warts on hand and forearm. A 51-year-old female with recalcitrant warts on hands for 2 years. Immunodeficiency was suspected but not detected.

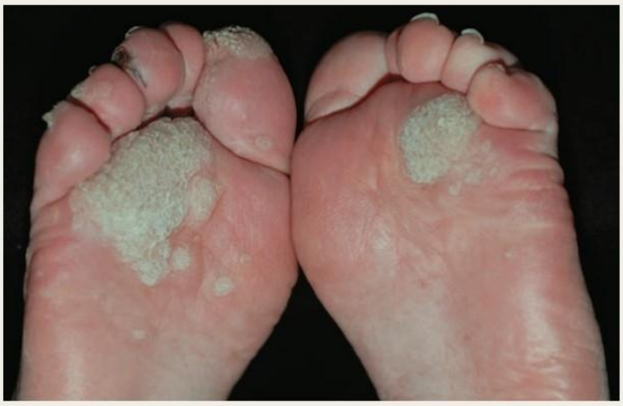

Figure 27-16. Verruca plantaris: plantar feet A 71-year-old male with chronic lymphatic leukemia. Large and painful on pressure, warts are seen on the plantar feet and toes. Multiple warts were also present on the fingers. After many failed therapeutic modalities, he was successfully treated with electron beam radiation.

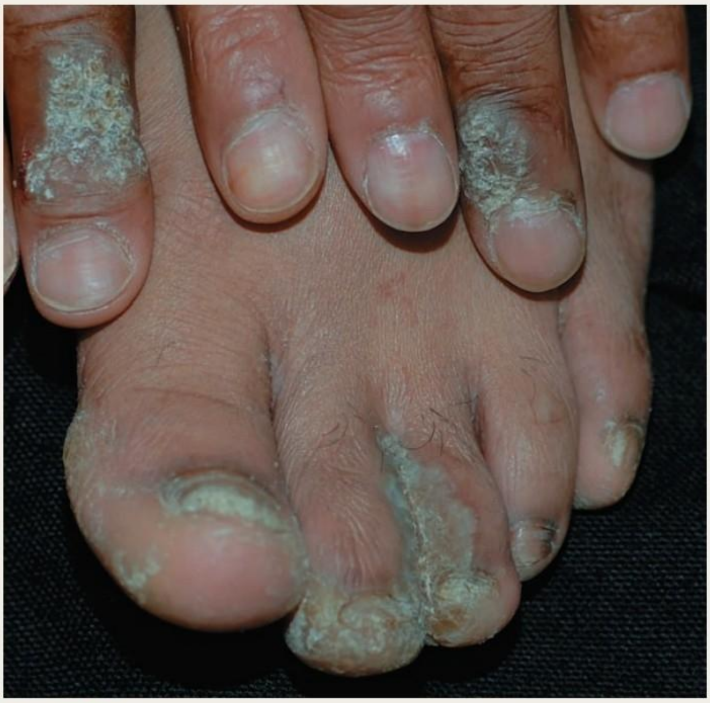

Figure 27-17. Extensive verrucae A 49-year-old male with HIV disease has confluent warts on the hands and feet. The large warts on opposing toes are referred to as “kissing warts.”

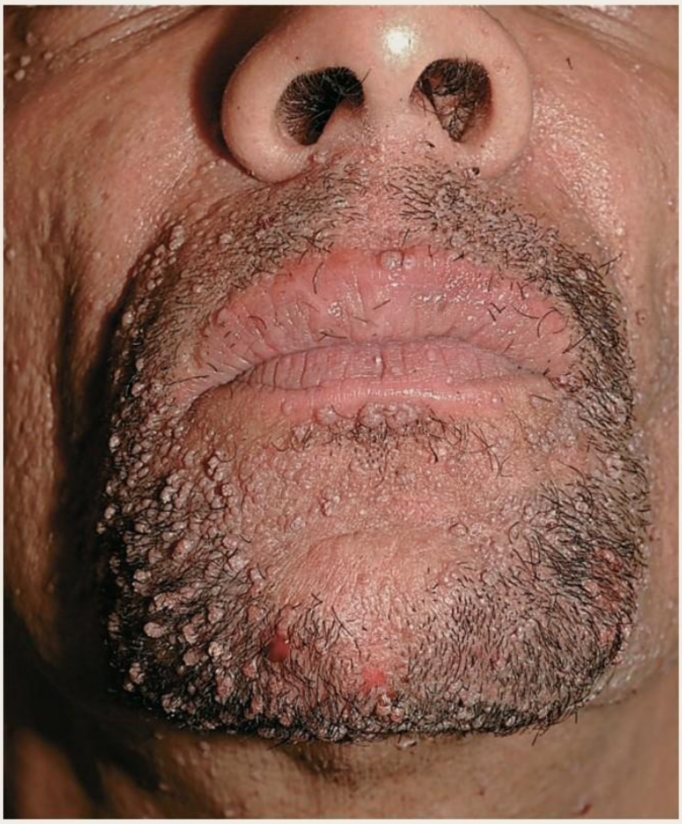

Figure 27-19. Filiform and flat warts A 38-year-old male with HIV disease has a confluence of lesions on face and beard area. Lesions resolved after successful antiretroviral therapy.

are usually to be avoided because the natural history of cutaneous HPV infections is for spontaneous resolution in months or a few years. Plantar warts that are painful because of their location warrant more aggressive therapies.

Patient-Initiated Therapy. Minimal cost; no/minimal pain.

For Small Lesions. 10–20% salicylic acid and lactic acid in collodion.

For Large Lesions. 40% salicylic acid plaster for 1 week, then application of salicylic acid–lactic acid in collodion.

Imiquimod Cream. At sites that are not thickly keratinized, apply half-strength three times per week. Persistent warts may require occlusion. Hyperkeratotic lesions on palms/soles should be debrided frequently; Imiquimod used alternately with a topical retinoid such as tazarotene topical gel may be effective.

Hyperthermia for Verruca Plantaris. Hyperthermia with hot water [45°C (113°F)] immersion for 20 minutes or three times weekly for up to 16 treatments is effective in some patients.

Clinician-Initiated Therapy. Costly, painful.

Cryosurgery. If patients have tried home therapies and liquid nitrogen is available, light cryosurgery using a cotton-tipped applicator or cryospray, freezing the wart and 1–2 mm of surrounding normal tissue for approximately 30 seconds, is quite effective. Freezing kills the infected tissue but not HPV.

Cryosurgery is usually repeated about every 4 weeks until the warts have disappeared. Painful.

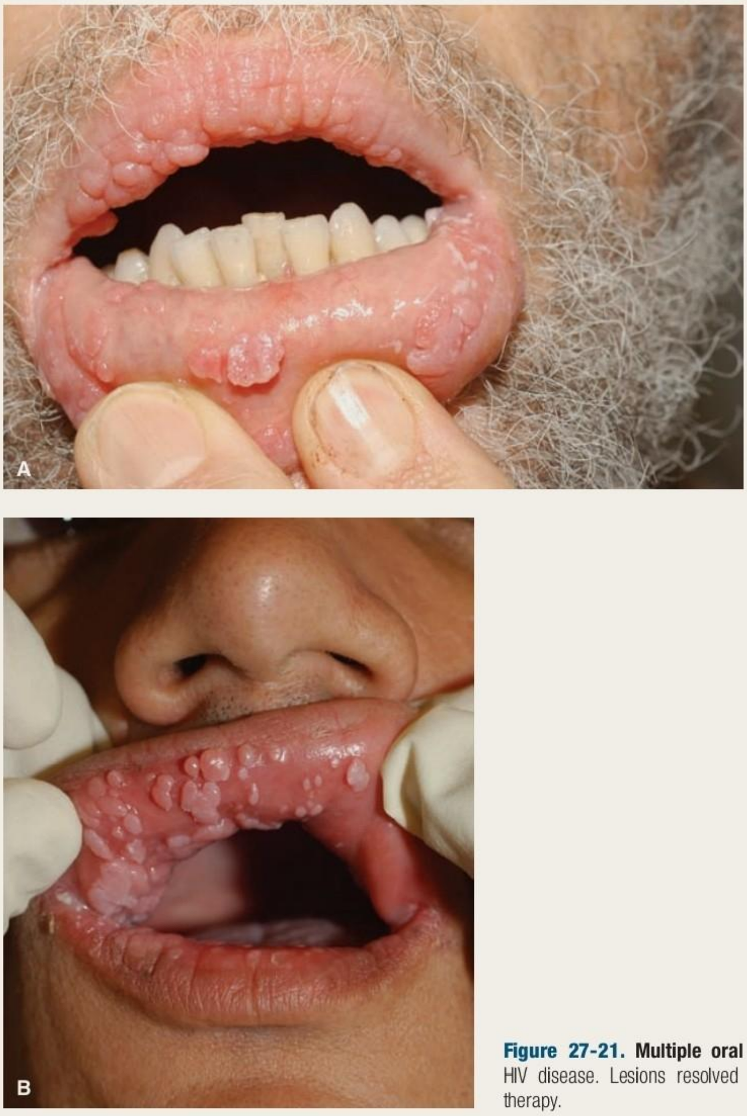

Figure 27-21. Multiple oral condylomata in HIV disease. Lesions resolved with antiretroviral therapy.

gesting leukocytoclastic vasculitis. Rash resolves with desquamation over 1–2 weeks. Profound fatigue persists for several weeks after recovery.

Differential Diagnosis

Other arborviral infection such as chikungunya and viral exanthems. Disease with local prevalence: typhoid, malaria, leptospirosis,

Consider diagnosis in travelers with febrile illness recently returned from endemic areas. During febrile phase, detection of viral nucleic acid in serum diagnostic. IgM seroconversion between paired samples is confirmatory finding.

Treatment

Symptomatic supportive therapy (http://www.cdc.gov/dengue/).

Herpes Simplex Virus Disease ICD-9: 054 ◦ ICD-10: B00 ☐ ● → ○

- Whether first symptomatic or recurrent may "typically" present clinically with grouped vesicles arising on an erythematous base on keratinized skin (Fig. 27-31) or mucous membrane. Most HSV infections are "atypical," with patch(es) of erythema, small erosions, fissures, or subclinical lesions that shed HSV.
- Following primary infection, HSV persists in sensory ganglia for the life of the patient, recurring with lessening in immunity.
- **Clinical Manifestation:**
  - In healthy individuals, recurrent infections are asymptomatic or minor, resolving spontaneously or with antiviral therapy.
  - With host defense defects, mucocutaneous lesions can be extensive, chronic, or disseminate to skin or viscera.

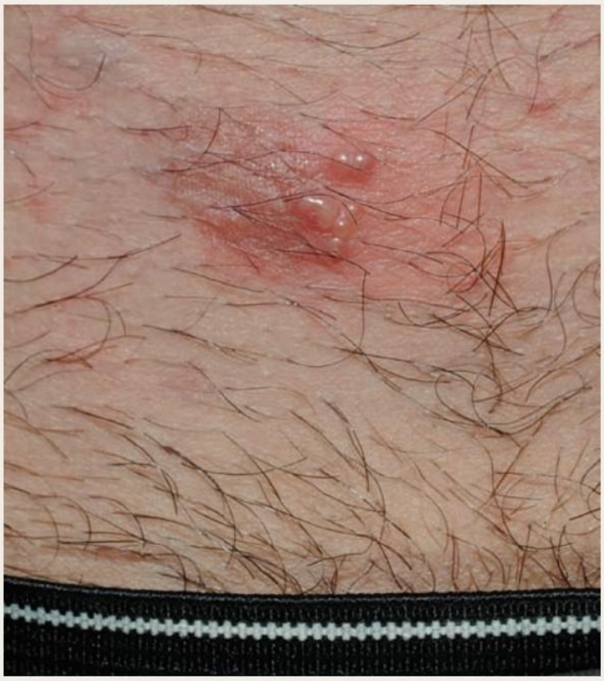

Figure 27-31. Herpes simplex: Typical lesion A 39-year-old male with lesion on the abdomen above the waist. Grouped vesicles on an erythematous base/plaque are seen. The lesion is recurrent.

Etiology and Epidemiology

Etiology. HSV-1 and HSV-2.

- Labialis: HSV-1 (80–90%), HSV-2 (10–20%).
- Urogenital: HSV-2 (70–90%), HSV-1 (10–30%).
- Herpetic whitlow: <20 years of age usually

sions. Usually skin–skin, skin–mucosa, mucosa–skin contact. Herpes gladiatorum transmitted by skin-to-skin contact in wrestlers.

Pathogenesis. Primary HSV infection occurs through close contact with a person shedding virus at a peripheral site, mucosal surface, or secretion. Transmission occurs via inoculation onto susceptible mucosal surface or break in skin (Fig. 27-32A). After exposure to HSV, the virus replicates in epithelial cells, causing

latency is established. Retrograde transport of HSV among nerves and establishment of latency are not dependent on viral replication in

Nongenital Herpes Simplex

- Nongenital HSV infection, whether primary or recurrent, is often asymptomatic.
- Lesions may present as group ve
erythematous base (Fig. 27-31)
erythematous plaque ± erosions

For genital HSV infection, see Sectio

Clinical Manifestation

Primary HSV Infection. Asymptomatic primary infection is common. Symptomatic primary HSV is characterized by *vesicles at the site of inoculation* (Fig. 27-34), and may be associated with regional lymphadenopathy, and systemic symptoms (fever, headache, malaise, myalgia). Primary

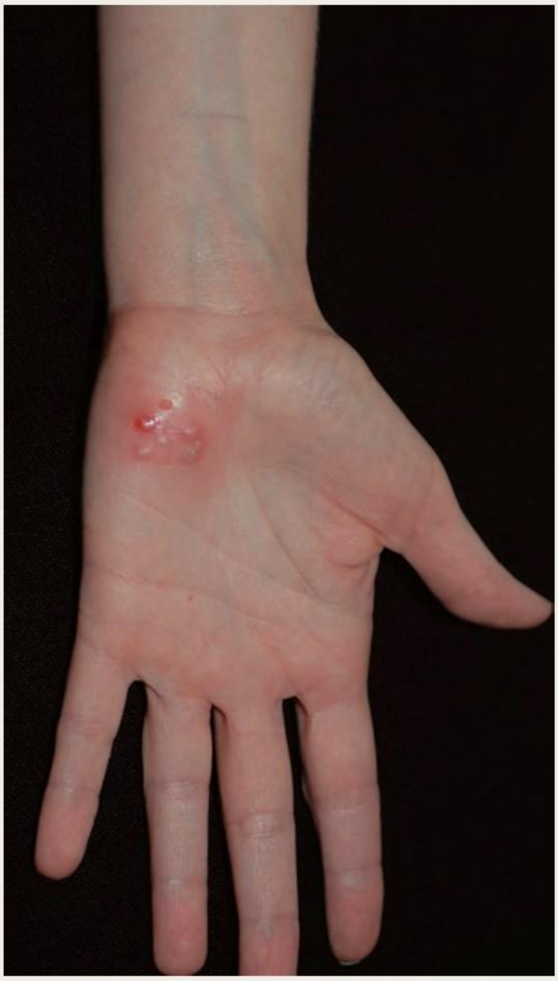

Figure 27-34. Herpes simplex: primary infection of the palm A 28-year-old female with a painful lesion on the palm for 3 days. A cluster of grouped pustules is seen on the palm. A red lymphangitis extends proximally on the wrist. The axillary lymph nodes were tender and enlarged. HSV-2 was detected on DFA. No antibodies to HSV-1 or -2 were detected, thus a primary infection.

herpetic gingivostomatitis is tl
symptom complex accompany
infection in children. Primary h
nitis is seen most often in young
Section 30).

Erythematous papules that q
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fragile, rupturing easily, to for
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Recurrent Herpes. Prodrome
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intraoral HSV is rare.

Trigeminal Nerve HSV Infection

- *Perioral infection.* Recurrent
cold sores are common (Fi
preceded by prodromal syn
pain, burning sensation,
recurrences may complica
ing surgery.
- *Ocular infections.* Recurren
major cause of corneal sca
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ommended.
- *Herpetic facial paralysis.* Reac
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HSV-1 shedding detected in

lymphadenopathy, and systemic symptoms (fever, headache, malaise, myalgia). Primary

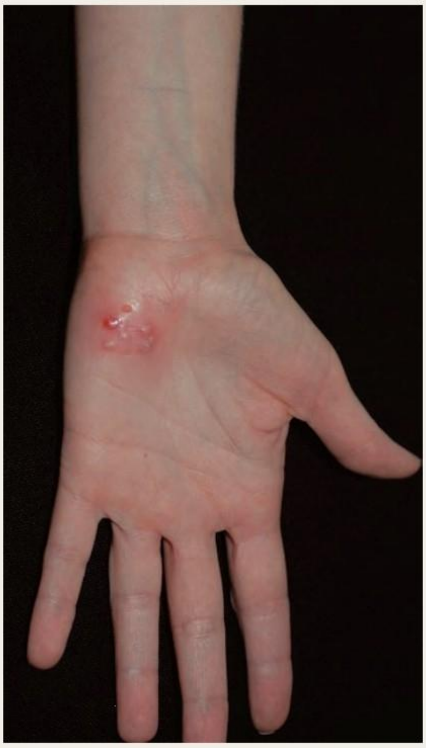


Figure 27-34. Herpes simplex: primary infection of the palm A 28-year-old female with a painful lesion on the palm for 3 days. A cluster of grouped pustules is seen on the palm. A red lymphangitis extends proximally on the wrist. The axillary lymph nodes were tender and enlarged. HSV-2 was detected on DFA. No antibodies to HSV-1 or -2 were detected, thus a primary infection.

grouped vesicles, and pustules occur at the site of inoculation (Fig. 27-34). Vesicles are often fragile, rupturing easily, to form *erosions* as the overlying epidermis sloughs. The most common sites of primary HSV infection are the mouth, anogenitalia, and hand/fingers. Erosions heal in 2–4 weeks, often with resultant postinflammatory hypo- or hyperpigmentation, uncommonly with scarring.

Regional Lymphadenopathy. May be tender.

Primary Herpetic Gingivostomatitis. Oral mucosa usually involved only in primary HSV infection with vesicles that quickly slough to form erosions (Fig. 27-35) at any site in the oropharynx: scanty to numerous. Gingival erythema, edema, and tenderness, edema. Severe pain. Perioral facial involvement with vesicles and erosions common.

Recurrent Herpes. Prodrome of tingling, itching, or burning sensation usually precedes any visible skin changes by 24 hours. Systemic symptoms are usually absent. Grouped vesicles on erythematous base that evolve to erosions and crusts (Fig. 27-36A–D). Recurrent intraoral HSV is rare.

Trigeminal Nerve HSV Infections

- *Perioral infection.* Recurrent facial herpes or cold sores are common (Fig. 27-36). Often preceded by prodromal symptoms (tingling, pain, burning sensation, itching). Severe recurrences may complicate laser-resurfacing surgery.
- *Ocular infections.* Recurrent keratitis is a major cause of corneal scarring and visual loss, Continuous suppression therapy is recommended.
- *Herpetic facial paralysis.* Reactivation of geniculate ganglion infection implicated in pathogenesis of idiopathic facial palsy (Bell palsy). HSV-1 shedding detected in 40% of cases.

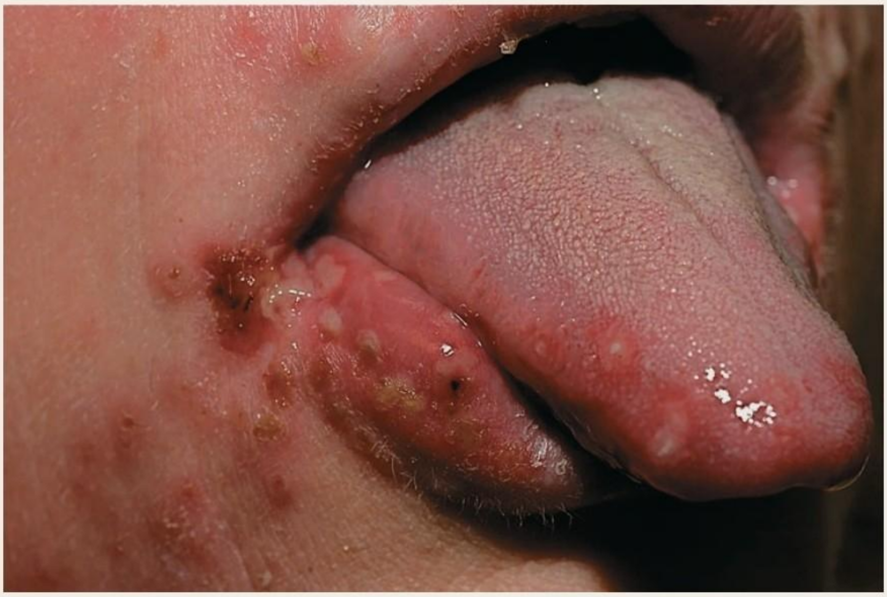


Figure 27-35. Herpes simplex: primary infection with gingivostomatitis A 43-year-old female with history of atopic dermatitis. Multiple, very painful erosions on the lower perioral skin, lips, and tongue. Tzanck smear was positive. HSV-1 detected on DFA. Methicillin-sensitive *S. aureus* (MSSA) was isolated on bacterial culture (secondary infection of herpetic lesions). HSV infection recurred on the face but without oral involvement.

- *Herpes gladiatorum.* Transmission occurs during contact sports (wrestling, rugby, football). Also occurs in cervical or lumbosacral dermatomes.

asymptomatic HSV reactivation/shedding from genital mucosa.

Complications of HSV Infections of Peripheral Sensory Nervous System

Cervical and Thoracic Sensory Nerve HSV Infections

- *Herpetic whitlow*. Infection of the tip of finger or thumb; uncommonly toe. Prior to "Universal Precautions," occurred in health-care professionals, especially dental personnel. Associated with painful neuritis in the affected finger (Fig. 27-37) and forearm.
- *HSV infection of the nipple*. Related to transmission of HSV from infant to mother during breast feeding.
- *HSV infections of the lumbosacral sensory nerves.* When lumbosacral ganglia become infected subsequent to anogenital herpes, recurrent lesions can occur on genitalia as well as buttocks, thighs, and perianal mucosa. Perianal herpes does not necessarily imply direct anal inoculation of HSV. Herpes in the sacral dermatome may be accompanied by
- ***Eczema herpeticum.*** Usually follows autoinoculation of HSV (most commonly orolabial herpes) to atopic dermatitis (see "Herpes Simplex Virus: Widespread Cutaneous Infection Associated with Cutaneous Immunocompromise," below p. 668).
- *S. aureus secondary infection.* Often occurs with eczema herpeticum.
- *Erythema multiforme.* In some individuals with recurrent HSV infections, erythema multiforme may occur with each recurrence (Fig. 27-38; see "Erythema Multiforme," Section 14).

General Findings. Fever may be present during symptomatic primary herpetic gingivostomatitis.
Regional Lymphadenopathy. Nonfluctuant, tender; usually unilateral.
CNS. Signs of aseptic meningitis: headache, fever, nuchal rigidity, CSF pleocytosis with normal sugar content, and positive HSV CSF culture.

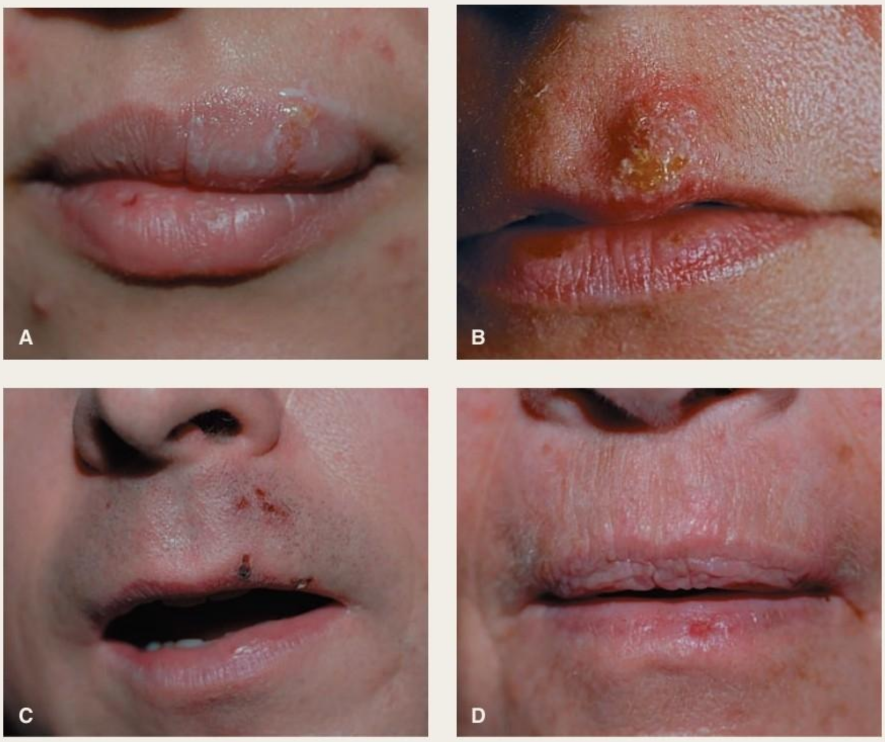


Figure 27-36. Herpes labialis: recurrent herpes labialis (A) Edematous lateral upper lip 24 hours after onset of tingling sensation. **(B)** Grouped vesicles on moustache area 48 hours after onset of symptoms. **(C)** Crusted erosion on upper lip and moustache area 7 days after onset of symptoms. **(D)** Painful erosion on the lower lip for 5 weeks in a 66-year-old female with severe dermatoheliosis and actinic cheilitis. The diagnosis was made on lesional biopsy.

Differential Diagnosis

Primary Intraoral HSV Infection. Aphthous stomatitis, hand foot and mouth disease, herpangina, erythema multiforme.
Recurrent Lesion. Fixed drug eruption.

Laboratory Examinations

See p. 662.

Diagnosis

Clinical suspicion confirmed by Tzanck smear, viral culture, or antigen detection DFA.

Course

Recurrences of HSV tend to become less frequent in time. Eczema herpeticum may

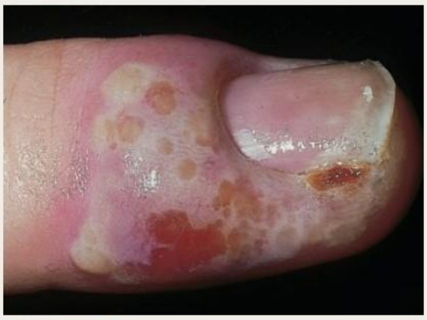


Figure 27-37. Herpes simplex virus infection: herpetic whitlow A 19-year-old male with painful finger lesions for 3 days. Painful, grouped, confluent vesicles on an erythematous edematous base of the distal finger were the first (and presumed primary) symptomatic infection.

Laboratory Examinations

See p. 662.

Diagnosis

Clinical suspicion confirmed by Tzanck smear, viral culture, or antigen detection DFA.

Course

Recurrences of HSV tend to become less frequent in time. Eczema herpeticum may complicate various dermatoses. Patients with host defense defects may experience cutaneous dissemination of HSV, systemic dissemination of HSV, and chronic herpetic ulcers (see also Chronic Herpetic Ulcers). Erythema multiforme (see Section 14) may complicate each recurrence of herpes, occurring 1–2 weeks after an outbreak.

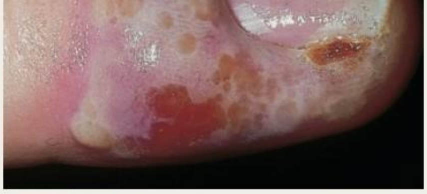


Figure 27-37. Herpes simplex virus infection: herpetic whitlow A 19-year-old male with painful finger lesions for 3 days. Painful, grouped, confluent vesicles on an erythematous edematous base of the distal finger were the first (and presumed primary) symptomatic infection.

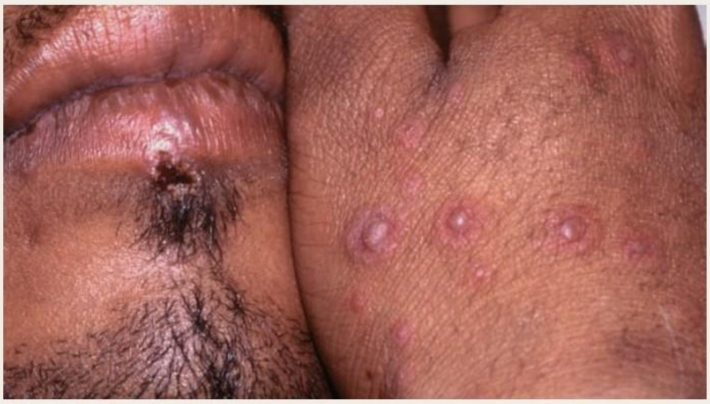


Figure 27-38. Herpes simplex virus infection: recurrent erythema multiforme A 31-year-old male with recurrent herpes labialis and disseminated lesions. Recurrent herpes labialis on the lower lip and IRIS-like edematous papules on the dorsum of the hand.

Treatment

See p. 662.

Neonatal Herpes Simplex ICD-9: 771.2 ∘ ICD-10: P35.2 ◧ ◐ → ○

- **Risk factors** for neonatal HSV infection: primary genital herpes in mother at time of delivery, absent maternal anti-HSV antibody, procedures on fetus, father with HSV infection.
- **Etiology.** The majority of infections are caused by HSV-2; HSV-1 is more virulent in the newborn and associated with higher morbidity and mortality
- **Transmission.** In utero (<5%); intrapartum (85%); postnatal acquisition. Mother is the most common source of infection. There is usually no clinical indication of shedding at the time of delivery. Shedding also occurs from uterine cervix. Incubation period in neonate: 4–21 days.
- **Demography.** Ninety-five percent of newborns with HSV infection contract it during labor and delivery (Figs. 27-39 and 27-40). Risk of transmission of HSV-2 from mother to newborn higher when primary infection occurs in third trimester. Maternal antibodies transferred to fetus and protect against fetal infection.

Clinical Manifestation

Skin, Eyes, Mouth Herpes Simplex. Localized infection. Vesicles and erosions on skin, eyes, mouth. Occurs at sites of trauma such as fetal scalp electrodes, extractors (vacuum and forceps), and circumcision. Margin of eyes and nasopharynx,

Disseminated Herpes. Disseminated infection. ±Vesicles, erosions. Hepatitis, pneumonitis, disseminated intravascular coagulation. Difficult to diagnose in that up to 70% of infants have no mucocutaneous lesions.

CNS Infection. ± Vesicles, erosions. Encephalitis. Presentation: seizures, tremors, lethargy, unstable temperature, irritability, feeding problem, bulging fontanelle.

Treatment

See p. 662.

disseminated intravascular coagulation. Diffi-

See p. 662.

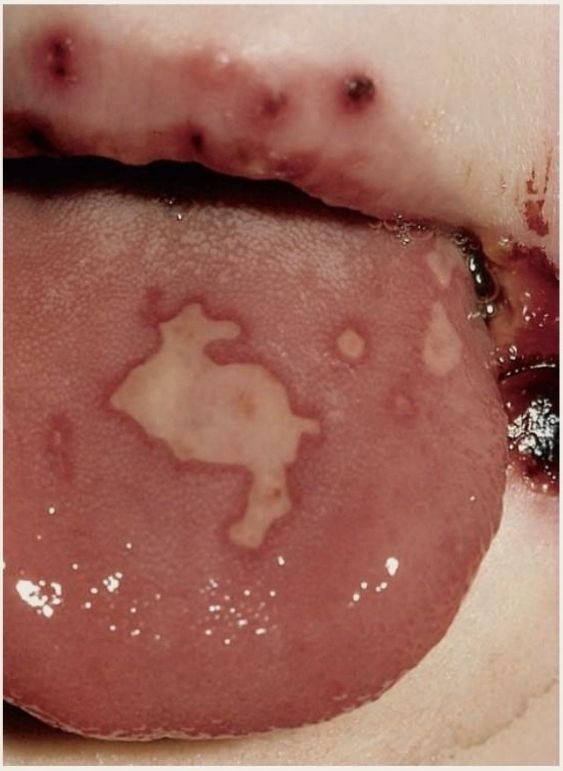


Figure 27-39. Herpes simplex in neonate Fever and skin lesion. *Vesicles* and crusted erosions on the upper lip and large geographic ulcerations of the tongue, i.e., herpetic gingivostomatitis.

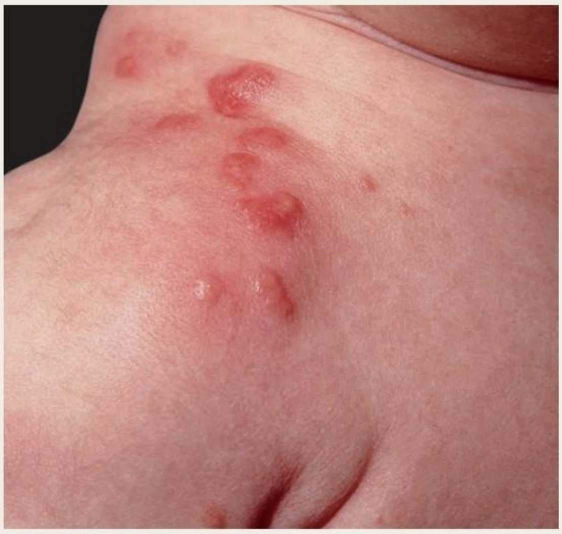


Figure 27-40. Herpes simplex virus infection: neonatal Neonate with skin lesion. Grouped and confluent vesicles with underlying erythema and edema on the shoulder, arising at the inoculation site.

Eczema Herpeticum ◧ ○

- HSV infects altered epidermis, most commonly atopic dermatitis causing eczema herpeticum. Other dermatoses subject to HSV infection include Darier disease, thermal burns, Hailey–Hailey disease, immunobullous disease, ichthyosis vulgaris, and cutaneous T cell lymphoma.
- Epidemiology. HSV-1 > HSV-2. More common in children. May be transmitted from parental herpes labialis to child with atopic dermatitis, especially if erythrodermic.

vesiculation may occur. Common sites: face, neck, and trunk.

temic dissemination can occur, especially with host defense defects. For treatment, see p. 662.

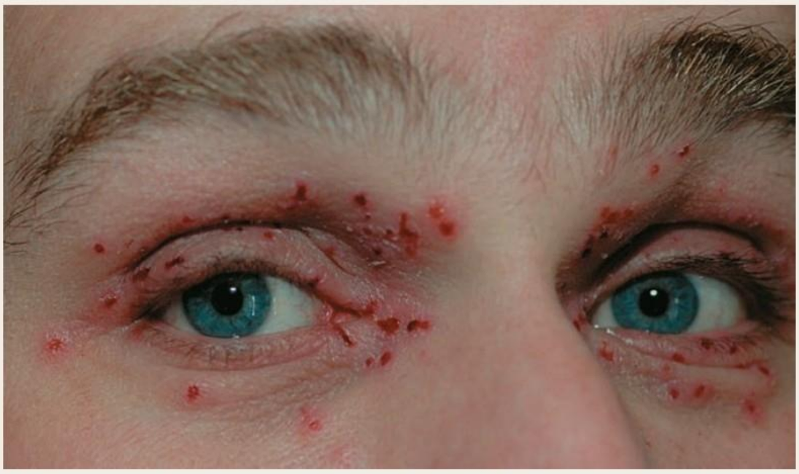
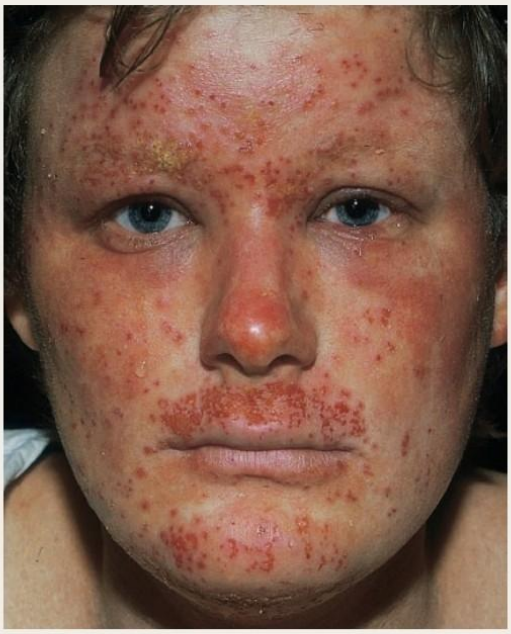


Figure 27-41. Herpes simplex: eczema herpeticum A 36-year-old male with recurrent periorbital painful crusted erosions and atopic dermatitis. Small-crusted erosion on the eyelids. DFA detected HSV-1. Bacterial culture reported MSSA. The herpetic infection had not affected the cornea.

Figure 27-42. Herpes simplex: extensive eczema herpeticum Confluent and discrete crusted erosions associated with erythema and edema of the face of a female with atopic dermatitis.



Herpes Simplex with Host Defense Defects ◧ ◐ → ○

- In persons with host defense defects, herpes simplex may present as extensive local involvement, chronic herpetic ulcers, or skin disease associated with systemic HSV infection.
- **Host Defense Defects.** HIV disease, leukemia/lymphoma, bone marrow transplantation, chemotherapy for solid organ or BMT, autoimmune diseases, malnutrition.
- **Pathogenesis.** After HSV viremia, disseminated cutaneous or visceral disease may occur. Factors determining whether severe localized disease, cutaneous dissemination, or visceral dissemination

Primary Herpetic Infection. Local infection may be widespread on the face (Fig. 27-43), oropharynx, and anogenital region with initial vesiculation followed by crusted erosions.

antiviral therapy (Fig. 27-45) (acyclovir, valacyclovir, famciclovir) are usually caused by acyclovir-resistant HSV.
Oropharyngeal Ulcers. Large ulcerations occur

oropharynx, and anogenital region with initial vesiculation followed by crusted erosions. Without antiviral therapy, lesions may persist to become chronic herpetic ulcers.

Recurrent Herpes Simplex. With advanced HIV disease especially, mucocutaneous disease can be severe: fingers with herpetic whitlow (Fig. 27-44A), oropharyngeal ulcers (Fig. 27-44B), esophageal ulcers, and anorectal ulcers. Systemic dissemination (Fig. 27-46) can occur from these sites, associated visceral HSV infection. Recurrent herpes simplex is manifested as

acyclovir-resistant HSV.

Oropharyngeal Ulcers. Large ulcerations occur on the tongue, hard palate, gingivae. Linear ulcerations occur on the tongue (Fig. 27-44B).

Esophageal Ulcers. Usually associated with oropharyngeal herpetic ulcer. Esophagoscopy demonstrates mucosal erosions/ulceration.

Anogenital Ulcers. Acute ulceration of the vulva, penis, scrotum, and/or perineum may become chronic ulcers unless effectively treated. In individuals infected with acyclovir-resistant HSV, ulcerations do not respond to usual

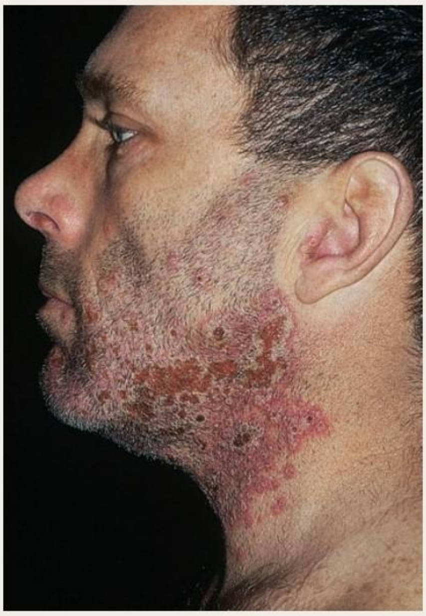


Figure 27-43. Herpes simplex: primary infection in HIV disease A 35-year-old male with HIV disease (CD4 cell count, 400/mL). Confluent vesicles and erosions with underlying erythema and edema (5–6-days duration) in the beard area. Gingivostomatitis and acute lymphadenopathy were also present, with onset of 5 days after orogenital sex.

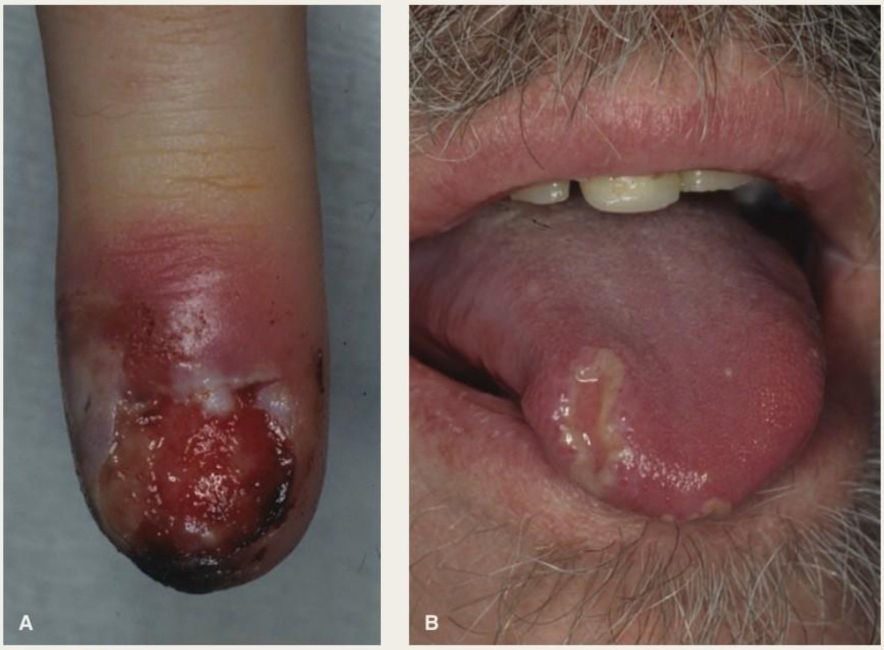


Figure 27-44. A 52-year-old male with advanced HIV disease had chronic herpetic ulcers on nares, finger, and tongue. **(A)** Herpetic whitlow with ulcer on the distal finger; nail had been avulsed by hand surgeon. **(B)** Chronic deep painful ulcer on the dorsolateral tongue.

antiviral therapies. Anal ulcers usually occur

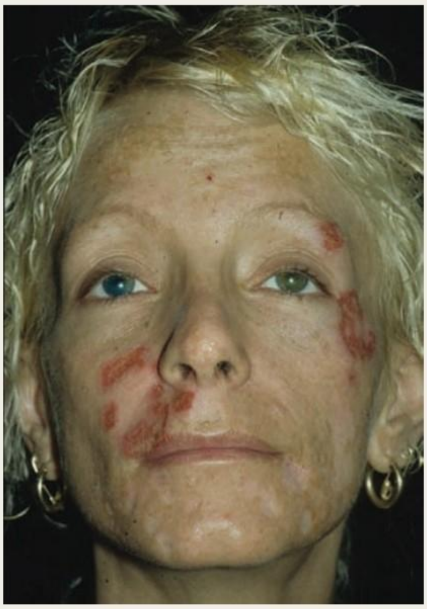


Figure 27-45. Herpes simplex: chronic herpetic ulcers A 40-year-old female with advanced HIV disease. Ulcers were caused by acyclovir resistant HSV, healed with foscarnet, but recurred.

antiviral therapies. Anal ulcers usually occur via enlargement of perianal ulcers. Herpetic proctitis: sigmoidoscopy shows friable mucosa and ulcerations.

Mucocutaneous Dissemination. Disseminated (nongrouped) vesicles and pustules often hemorrhagic with inflammatory halo; quickly rupture, resulting in "punched-out" erosions. Lesions may be necrotic and then ulcerate (Fig. 27-46).

General Examination. Widespread visceral involvement (liver, lungs, adrenals, GI tract, CNS) can occur in persons with severe host defense defects.

Differential Diagnosis

Chronic Herpetic Ulcers. Chronic VZV infection, wound infection, pressure ulcer

Anorectal Ulcers. HPV-induced SCC, Crohn disease

Mucocutaneous Dissemination. Varicella or disseminated herpes zoster (HZ), eczema herpeticum.

Diagnosis

Clinical suspicion confirmed by Tzanck smear, positive HSV antigen detection DFA, or isolation of HSV on viral culture.

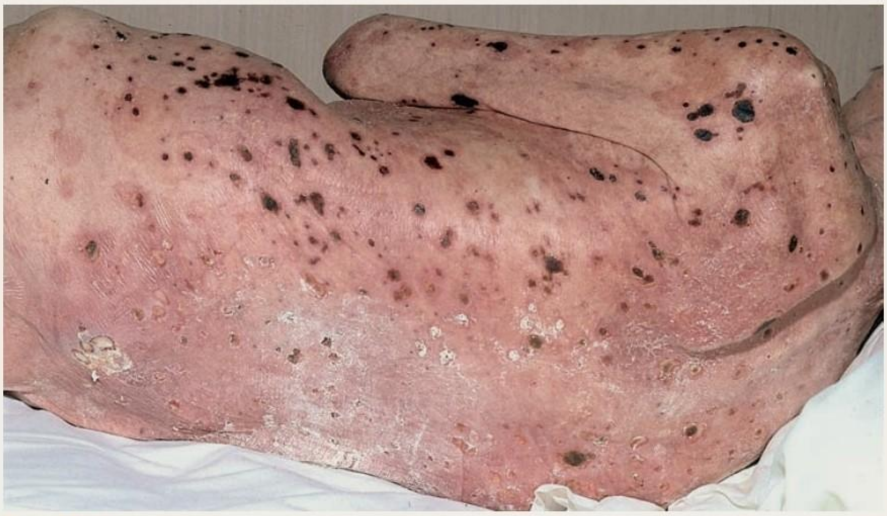


Figure 27-46. Disseminated herpes simplex 60-year old male with lymphoma. Disseminated erosions, ulcerations with hemorrhagic crusts on necrotic bases. Patients often have HSV visceral infection (lungs, liver, brain).

Course and Treatment

For treatment, see p. 662. In HIV disease, persons successfully treated with ART experience reduction in frequency and severity of HSV recurrences. Infection with acyclovir-resistant strains results in chronic, progressive ulcerations that persist and/or continue to enlarge despite oral and IV acyclovir treatment.

Varicella Zoster Virus Disease
ICD-9:052 ◦ ICD-10:B01

☐ ● → ◐

- **Varicella zoster virus** is a HHV that infects 98% of adults.

the neuron to the skin, where it erupts in a dermatomal pattern, i.e., HZ or *shingles*.

wheals and quickly evolve to *vesicles*, superficial and thin-walled with surrounding erythema. Vesicles rapidly evolve to pustules and *crusted*

blades, flanks, axillae, popliteal, and antecubital fossae. Density highest on trunk and face, less on extremities. Palms and soles usually spared.

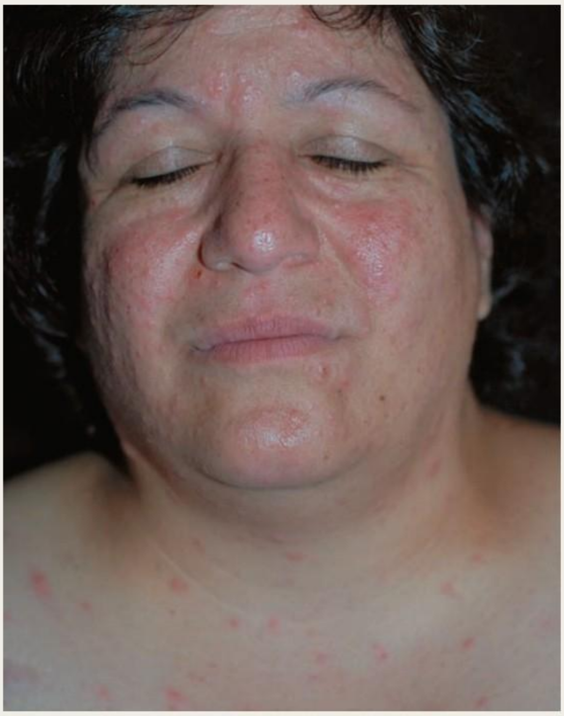


Figure 27-47. Varicella A 20-year-old female with pruritic eruption for 2 days. Multiple, pruritic, erythematous papules, vesicles on the face and neck. Several vesicles have evolved to crusted erosion. DFA detected VZV. No antibodies to VZV were detected.

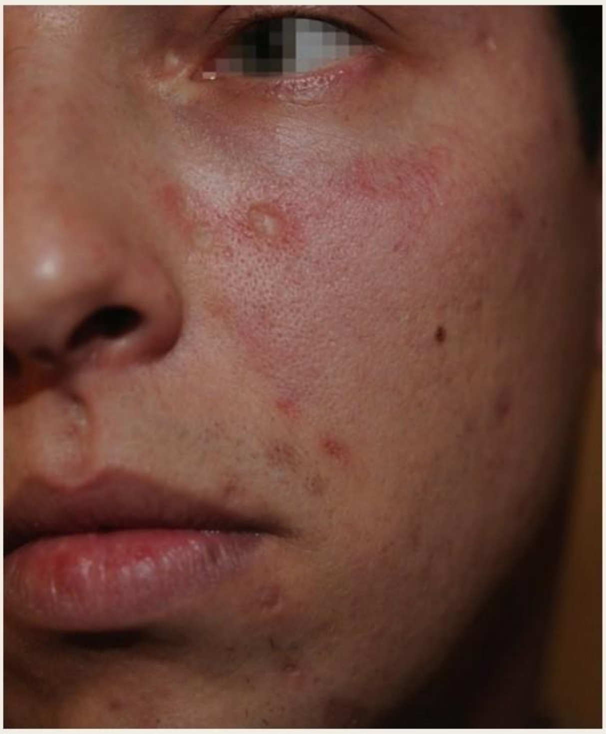


Figure 27-48. Post-varicella scars A 28-year-old male with punched out scars on face. Varicella, which had been severe, had occurred 6 months previously. These scars may improve with time but persist for life.

Mucous Membranes. Vesicles (not often observed) and subsequent shallow erosions (2–3 mm). Most common on palate. Less common on other mucosal sites.

General Examination. *VZV pneumonitis* occurs with increased frequency in adolescents and adults. *CNS* involvement with cerebellar ataxia and encephalitis can occur.

"Malignant" varicella occurs in persons with host defense defects. Pneumonitis, hepatitis, encephalitis, disseminated intravascular coagulation, and purpura fulminans may occur.

Course

The most common complication in children <5 years is secondary bacterial infection. *Varicella encephalitis* and *Reye syndrome* occur in children 5–11 years of age. Two percent of fetal varicella associated with maternal varicella in first trimester of pregnancy. *Fetal varicella syndrome*, characterized by limb hypoplasia, eye and brain damage, and skin lesions. Varicella in immunocompromised may be complicated by hepatitis, encephalitis, and hemorrhagic complications.

nerve occurs in about one-third of cases and

tis, erysipelas, and necrotizing fasciitis.

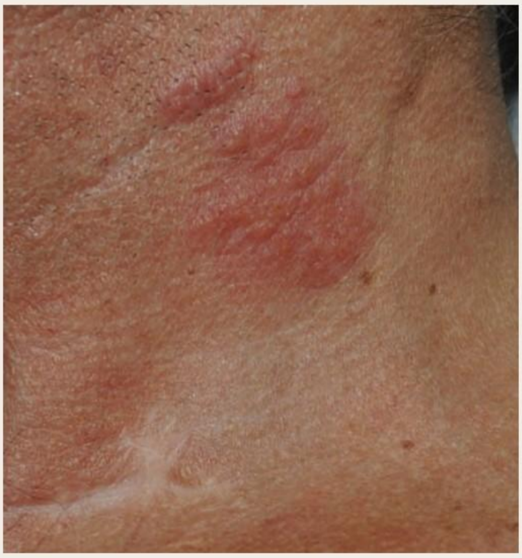
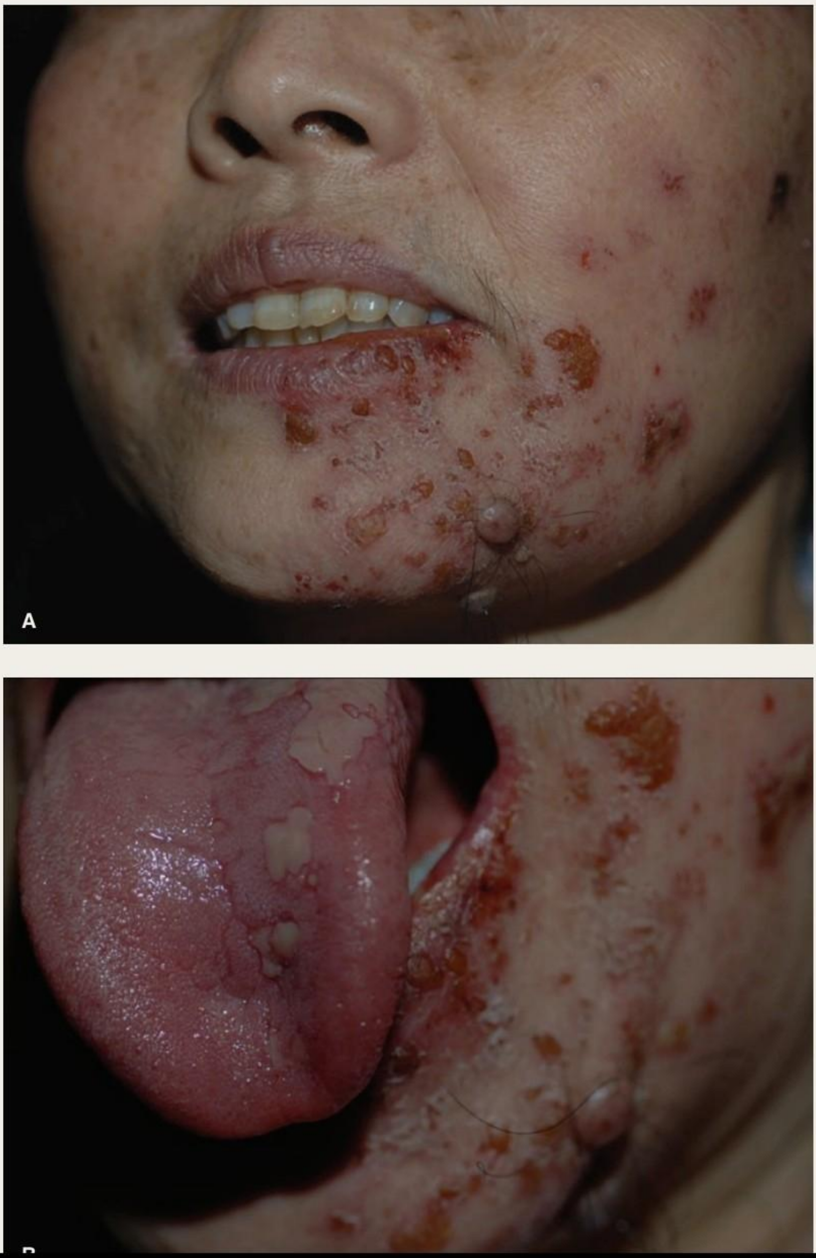


Figure 27-51. Herpes zoster A 65-year old male with scar from prior thyroid carcinoma surgery. Erythematous plaque with early vesiculation in left C-2 dermatome. This presentation is common and diagnosis often missed. The lesions was mildly pruritic.



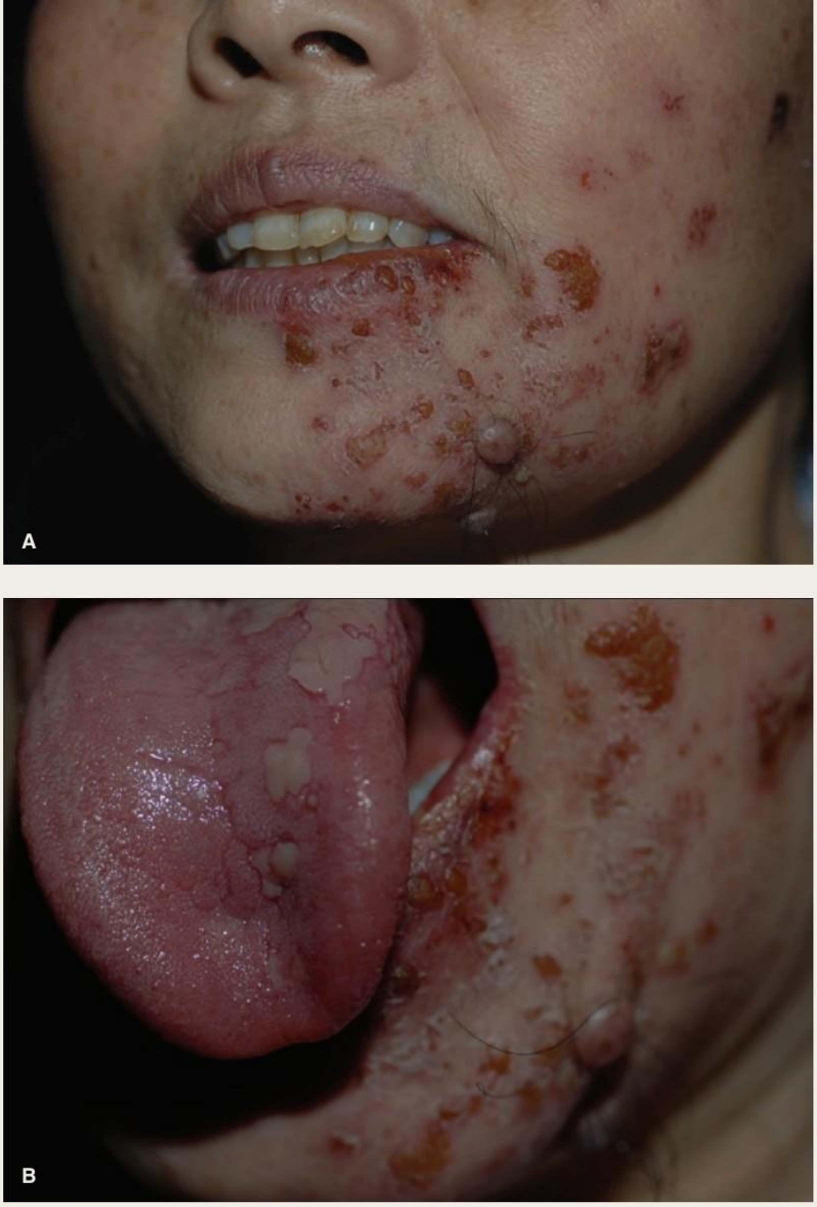
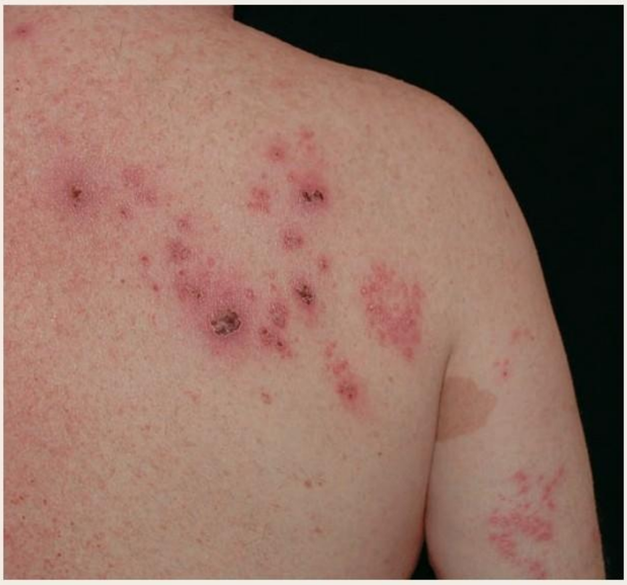
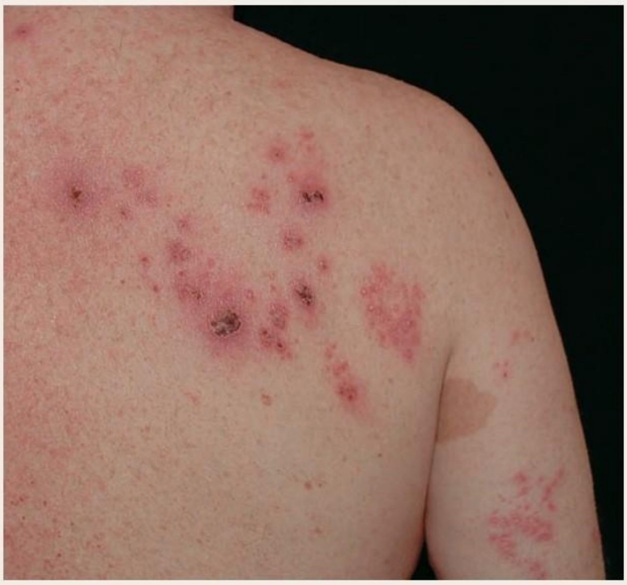


Figure 27-52. Herpes zoster A 67-year old Chinese female with dermatomal zoster in L-mandibular branch of the trigeminal nerve. Bullae, vesicle, and erosions are seen. **(A)** L-face. **(B)** Tongue with erosions and deviation associated with motor involvement. Other than flu-like symptoms, she was relatively free of symptoms.

Figure 27-53. Herpes zoster right T-2 distribution A 60-year-old male being treated with prednisone for eczema has painful lesion for 3 days. Dermatomal grouped and confluent vesicles on the R-back and arm.



branch of the trigeminal nerve. Bullae, vesicle, and erosions are seen. **(A)** L-face. **(B)** Tongue with erosions and deviation associated with motor involvement. Other than flu-like symptoms, she was relatively free of symptoms.



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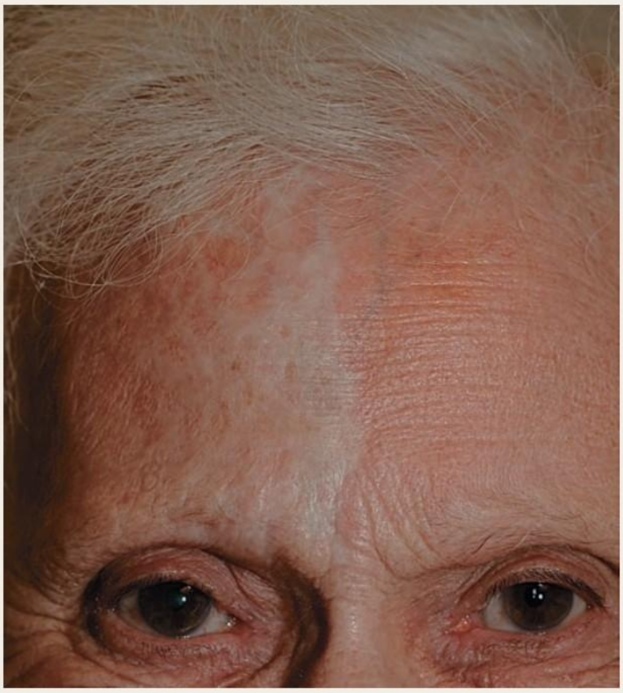


Figure 27-54. Herpes zoster: atrophic scar A 90-year-old female with a history of herpes zoster 14 years previously. Hypopigmented dermatomal (V1) scar is seen on the right forehead at the site of prior zoster.

Diagnosis

Prodromal Stage. Suspect zoster in older or immunocompromised with unilateral pain.

Active Vesiculation. Clinical findings usually

est incidence is in ophthalmic zoster. Does not appear to be more common in immune defects than in the general population.

Pain with HZ is associated with neural

Herpes zoster: severe dermatomal disease (Fig. 27-55)

Herpes Zoster with Cutaneous Dissemination. Variable numbers of vesicles or bullae are seen at any mucocutaneous site (Fig. 27-57). The condition thus appears clinically as zoster plus varicella.

or verrucous dermatomal lesions (Fig. 27-58).

Eye. Acute retinal necrosis occurs in the absence of apparent conjunctival or cutaneous involvement with subsequent loss of vision.

Visceral Dissemination. Encephalitis, polyneuritis, myelitis, vasculitis; pneumonitis; hepatitis; pericarditis/myocarditis; pancreatitis; enterocolitis.

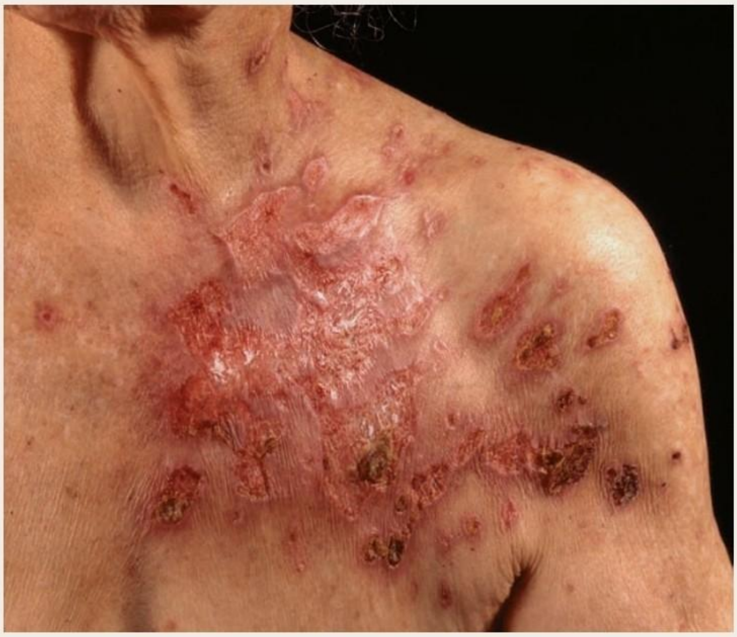


Figure 27-55. **VZV: necrotizing herpes zoster** Confluent, crusted ulcerations on an inflammatory base in several contiguous dermatomes in an elderly male with leukemia.

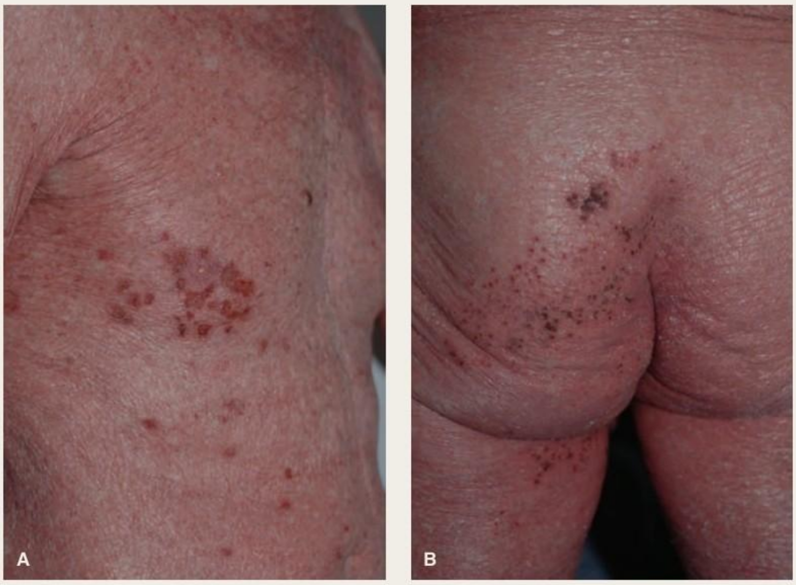
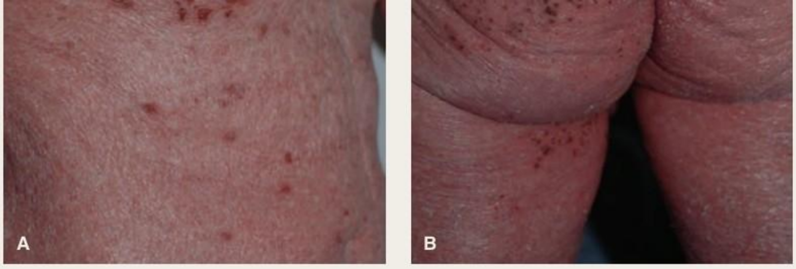


Figure 27-56. **Multidermal herpes zoster with host defense defect.** 72-year-old male with pityriasis rubra pilaris with erythroderma is being treated with prednisone and methotrexate. Multiple dermatomal erosions are seen on the chest and buttock with dissemination.



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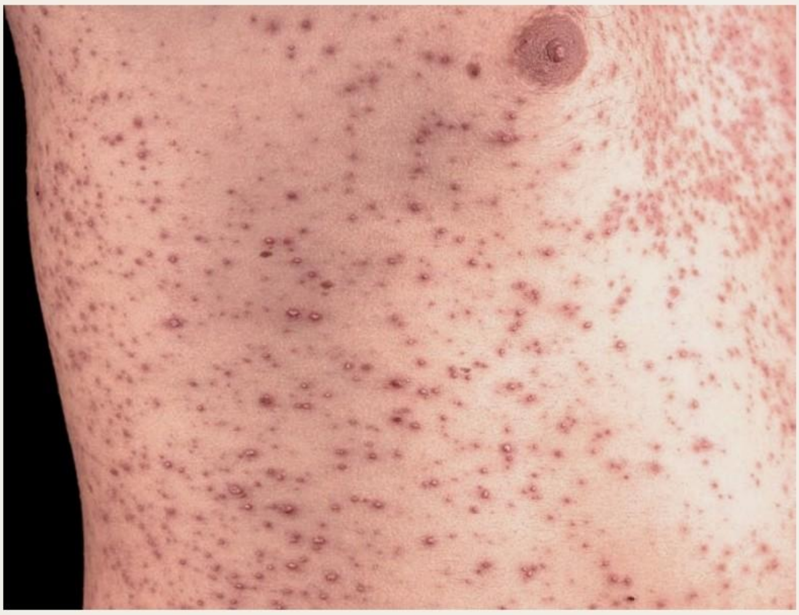

Figure 27-57. Varicella zoster virus infection: disseminated cutaneous, in an immunocompromised patient Hundreds of vesicles and pustules on erythematous bases of the trunk of a patient with lymphoma. Note the absence of grouping of lesions seen in herpes simplex or herpes zoster. The eruption is indistinguishable from varicella and must be differentiated from disseminated HSV infection.

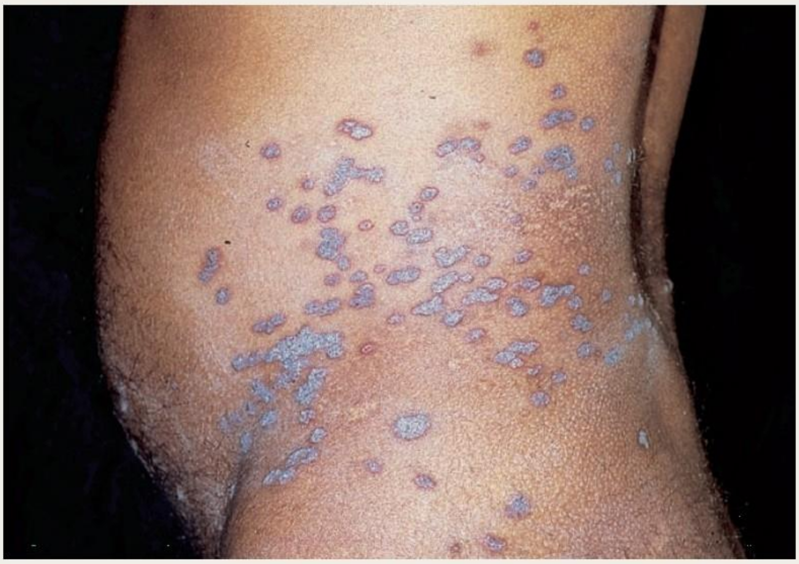

Figure 27-58. VZV: chronic zoster in HIV disease A 42-year-old male with advanced untreated HIV disease. Discrete and confluent hyperkeratotic papules/nodules in several contiguous dermatomes persistent for 2 years.

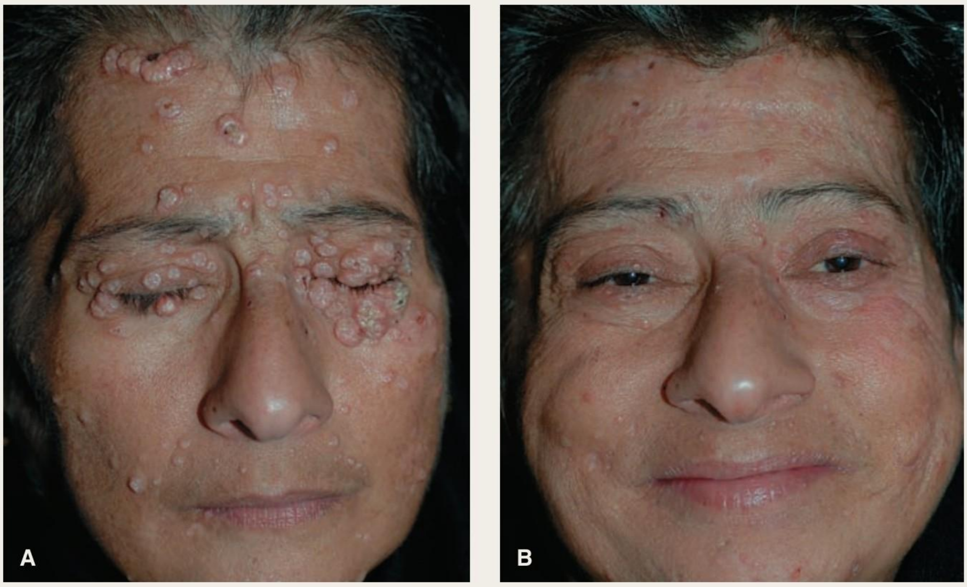

Figure 27-69. **Molluscum contagiosum, confluent** A 51-year-old female with advance HIV disease. **(A)** Extensive and confluent facial nodules were disfiguring. **(B)** Lesions resolved with electrodessication.

Human Papillomavirus Infection

Figure 28-1. Comparison of blacklegged, lone star, and dog ticks Black-legged or *Ixodes* nymphal ticks transmit *Borrelia burgdorferi* (Lyme disease) and other infections. Lone star ticks or *Amblyomma americanum* is the vector for anaplasmosis, tularemia, and Southern tick-associated rash illness. Dog or wood ticks, *Dermacentor variabilis*, transmit Rocky Mountain spotted fever and tularemia.

- Malaria, babesiosis.
- Filariasis, onchocerciasis (river blindness), loiasis

Clinical Manifestation

Erythematous macules. occur at bite sites and are usually transient.

Papular urticaria or urticarial papules persistent for >48 h (Fig. 28-2, Fig. 28-3); usually <1 cm; vesicle may for on papule. Large urticarial plaques may occur.

Bullous Lesions. Tense bullae with clear fluid on a slightly inflamed base (Fig. 28-4); excoriation results in large erosion.

Secondary Lesions. Excoriations of urticarial, papular, vesicular lesions common. Painful erosion may be secondarily infected with *Staphylococcus aureus*. Excoriated or secondarily infected lesions may heal with hyper- or hypopigmentation and/or raised or depressed scars, especially in more darkly pigmented individuals.

Systemic findings may occur associated with toxin or allergy to substance injected during bite. Many varied systemic infections can be injected during bite.

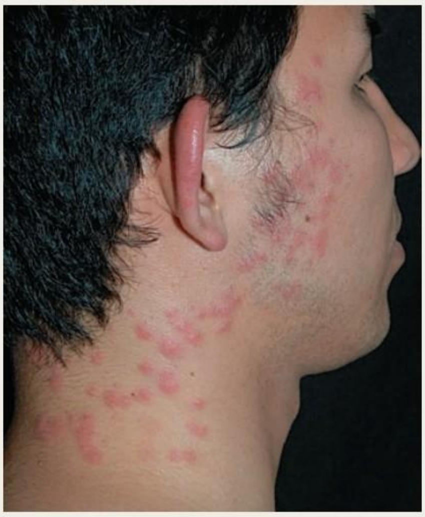

Figure 28-2. Papular urticaria A 21-year-old male awoke with multiple pruritic erythematous papules on exposed of face, neck, forearms, and hands. Bedding was heavily colonized with bedbugs.

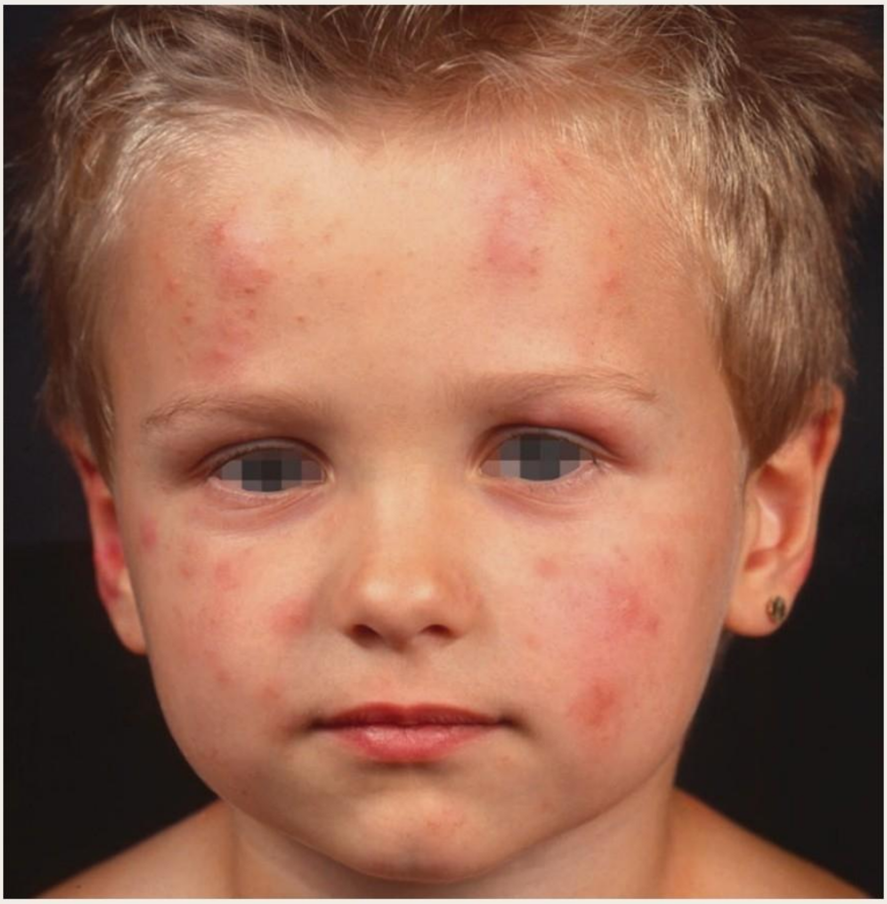

Figure 28-3. Papular urticaria A 6-year-old girl with multiple mosquito bites on face.

Clinical Variations by Arthropod

Mites. *Sarcoptes scabiei* causes infestation *scabies* (see Scabies). *Demodex folliculorum* and *D. brevis* live *in* human hair follicles and sebaceous glands, causing *demodicidosis* (see Fig. 28-15).

Food, fowl, grain, straw, harvest, and animal

species transmits *Rickettsia tsutsugamushi*, the cause of scrub typhus.

Dermatophagoides species of house dust mites are implicated in the pathogenesis of asthma and atopic dermatitis. Feed on desquamated human skin and other organic detritus, living in bedding, carpets, and furniture. Bodies and excreta may have a role in asthma and other allergies. Affected persons respond with production of IgE antibodies. *Fowl mites*,

Clinical Variations by Arthropod

Mites. *Sarcoptes scabiei* causes infestation *scabies* (see Scabies). *Demodex folliculorum* and *D. brevis* live *in* human hair follicles and sebaceous glands, causing *demodicidosis* (see Fig. 28-15).

Food, fowl, grain, straw, harvest, and animal mite bites cause papular urticaria.

Food Mites. Cheese, grain, mold mites can cause mild contact dermatitis: baker's or grocer's itch. *Straw mites.* Bites occur during harvest season causing dermatitis; straw itch. *Harvest mite:* Chiggers. Bites can cause dermatitis. One mites are implicated in the pathogenesis of asthma and atopic dermatitis. Feed on desquamated human skin and other organic detritus, living in bedding, carpets, and furniture. Bodies and excreta may have a role in asthma and other allergies. Affected persons respond with production of IgE antibodies. *Fowl mites.* Chicken, pigeons, etc. Bites cause papular urticaria on exposed sites. *Rat mites* cause painful bites and dermatitis and transmit endemic/murine typhus. *House mouse mite* is the vector for rickettsialpox. *Cheyletiella* spp. (dog and cat mites) bite pet owners causing pruritic lesions on forearms, chest, and abdomen. Canine sarcoptic mange (*S. scabiei* var. *canis*) and feline mange (*Notoedres cati*) cause a pruritic dermatosis in pet owners.

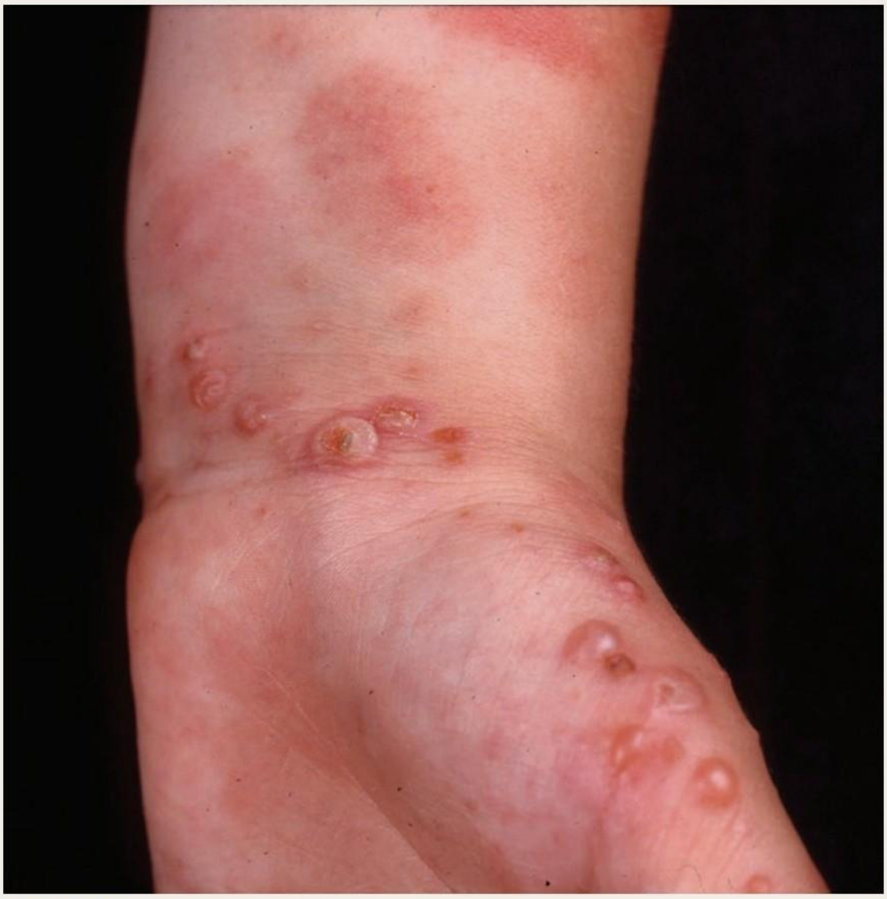

Figure 28-4. Bullous insect bite A 10-year-old child with bullous lesions on the ventral wrist and popular urticarial on the forearm.

Ticks. *Ticks* attach and feed painlessly. Secretions can produce local bite reactions (erythema), febrile illness, and paralysis. Blacklegged or *Ixodes* tick, lone star tick, and dog tick are vectors for diseases. Erythema migrans (Fig. 25-81), characteristic of primary Lyme disease or borreliosis, occurs at the bite site of an infected *Ixodes* tick that transmits *Borrelia burgdorferi*.

Lymphocytoma cutis (Fig. 25-82) also occurs at the site of bite of an infected *Ixodes* tick.

Spiders. *Brown recluse spider* bites can result in mild local urticarial reactions to full-thickness skin necrosis. Associated with a maculopapular exanthem, fever, headache, malaise, arthralgia, and nausea/vomiting. Most lesions diagnosed as brown recluse spider bites are bite reactions to other arthropods. *Widow spiders* inject a neurotoxin (α-latrotoxin) that produces bite site reactions as well as varying degrees of systemic toxicity.

Insects. *Pubic lice, head lice, body lice* papular urticaria, excoriations, secondary infections (see page 707).

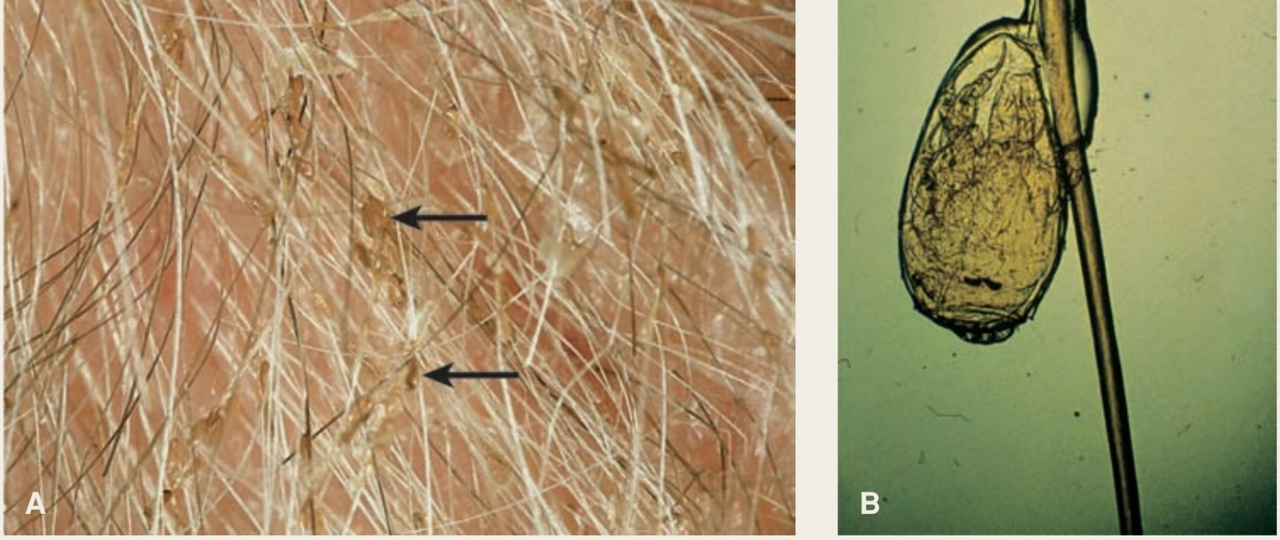

Figure 28-9. **Pediculosis capitis: nits (A)** Arrows: grayish-white egg capsules (nits) are firmly attached to the hair shafts, visualized with a lens. **(B)** Magnified, an egg with a developing head louse nymph attached to a hair shaft is seen.

from only a few to thousands. Nits are deposited by head lice on the hair shaft as it emerges from the follicle. With current infestation, nits

Differential Diagnosis

Small White Hair “Beads” Hair casts (inner root

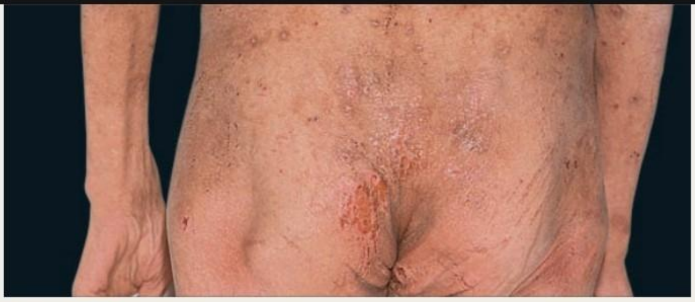

Figure 28-10. Pediculosis corporis A 60-year-old homeless male. Multiple lesions secondary to excoriations, prurigo nodularis, and lichen simplex chronicus. Lice and nits are seen in the seams of clothing (inset).

simplex, infection with *S. aureus*, and postinflammatory hyperpigmentation (Fig. 28-10). Scabies, pediculosis capitis, and *Pulex irritans* (the human flea) can coexist.

Differential Diagnosis

Atopic dermatitis, contact dermatitis, scabies, adverse cutaneous drug reaction.

Diagnosis

Lice and eggs are found in clothing seams.

Treatment

Decontamination of Clothing and Bedding. Hygiene measures.
Delousing. Pyrethrin, permethrin, malathion.

Pediculosis Pubis ICD-9: 132.2 ◦ ICD-10: B85.2 □ ● → ◐

- In infestation of hair-bearing regions by the crab or pubic lice.
- Most commonly inhabit the pubic area; hairy parts of the chest and axillae; upper eyelashes.
- Manifested clinically by mild-to-moderate pruritus, papular urticaria, and excoriations.

Etiology and Epidemiology

Phthirius pubis, the crab or pubic louse. Size 0.8–1.2 mm. First pair of legs vestigial; other two clawed (Fig. 28-11). Life span 14 days. Female lays 25 ova. Nits incubate for 7 days; nymphs mature over 14 days. Mobility: adults can crawl 10 cm/day. Prefer a humid environment; tend not to wander. Infestation most common in young males. Transmission during close physical contact: sharing bed. May coexist with another sexually transmitted diseases.

Clinical Manifestation

Often Asymptomatic. Mild-to-moderate pruritus for months. With excoriation and secondary infection, lesions may become tender and be associated with enlarged regional.
Infestation. *Lice* appear as 1- to 2-mm, brownish-gray specks (Fig. 28-12, Fig. 28-13) in hairy areas involved. Remain stationary for days; mouthparts embedded in skin; claws grasping a hair on either side. Usually few in number. *Nits* attached to hair appear as tiny white-gray specks (Fig. 28-13). Few to numerous. Eggs at hair–skin junction indicate active infestation. *Infestation* most common in pubic and axillary areas; also, perineum, thighs, lower legs, trunk, periumbilical. In children, eyelashes (Fig. 28-13) and eyebrows may be infested without pubic involvement. Maculae cœrulea most common on lower abdominal wall, buttocks, and upper thighs.

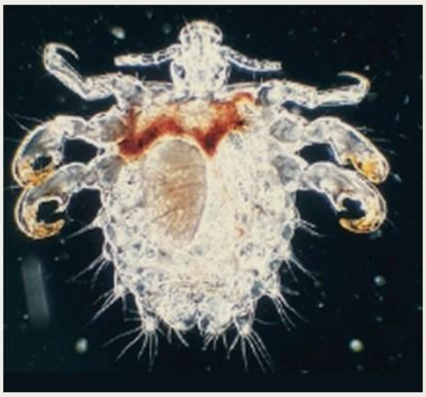

Figure 28-11. Crab louse Adult female with an egg developing within her body.

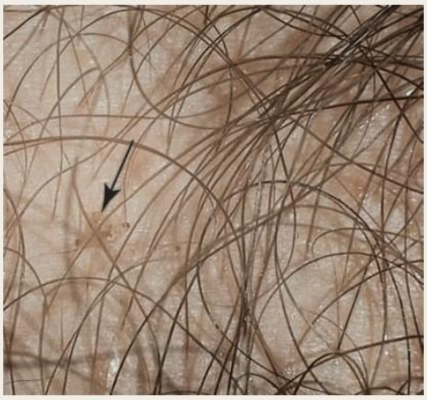

Figure 28-12. Crab louse Crab louse (arrow) on the skin in the pubic region.

adults in 14 days.

Habitat: live in seams of clothing; can survive without blood meal for up to 3 days. Attaches to body hairs to feed. Risk factors for infestation include poverty, war, natural disasters, indigence, homelessness, and refugee-camp populations.

Body Lice as Vectors of Disease. Body lice transmit many infectious agents while feed-

Infestation. Lice and nits are found in cloth seams (Fig. 28-10). Lice grab on to body h to feed.

Reactions to Bites. Bite reactions such as po lar urticarial (Fig. 28-10) are similar to those head lice. Changes secondary to rubbing scratching include excoriations, eczema, lic

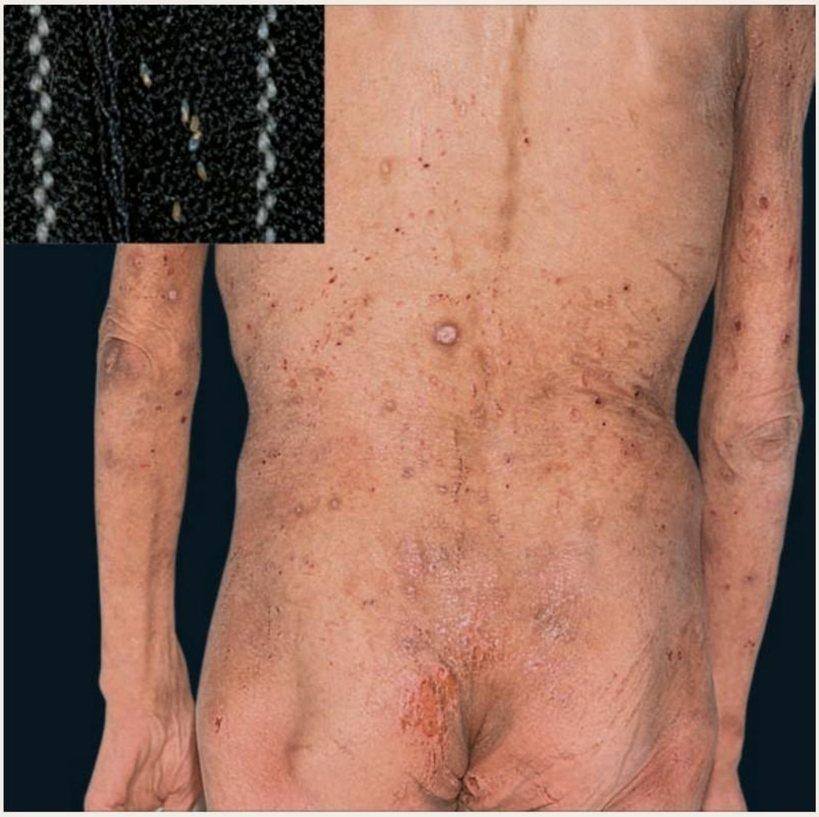

Figure 28-10. Pediculosis corporis A 60-year-old homeless male. Multiple lesions secondary to excoriations, prurigo nodularis, and lichen simplex chronicus. Lice and nits are seen in the seams of clothing (inset).

simplex, infection with *S. aureus*, and postinflammatory hyperpigmentation (Fig. 28-10). Scabies, pediculosis capitis, and *Pulex irritans* (the human flea) can coexist.

Differential Diagnosis

Atopic dermatitis, contact dermatitis, scabies, adverse cutaneous drug reaction.

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Delousing. Pyrethrin, permethrin, malathio

Pediculosis Pubis ICD-9: 132.2 ∘ ICD-10: B85.2 ☐ ● → ◐

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Etiology and Epidemiology

Phthirius pubis, the crab or pubic louse. Size 0.8

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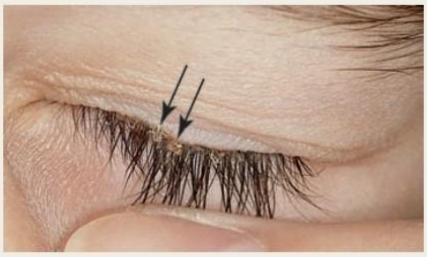

Figure 28-13. Crab lice in eyelashes A 10-year-old child. Crab lice (arrows) and nits on the upper eyelashes of a child; this was the only site of infestation.

Skin Lesions. *Papular urticaria* (small erythematous papules) at sites of feeding, especially periumbilical (Fig. 28-14). *Changes secondary* to rubbing lichenification and excoriations. Secondary *S. aureus infection*. *Maculae ceruleae* (*taches bleues*) are slate-gray or bluish-gray macules 0.5–1 cm in diameter, nonblanching. With eyelid infestation, serous crusts may be present along with lice and nits; occasionally, edema of eyelids with severe infestation.

With secondary impetiginization, regional lymphadenopathy.

Differential Diagnosis

Atopic dermatitis, seborrheic dermatitis, tinea cruris, molluscum contagiosum, scabies. These disorders may coexist with crab louse infestation.

Diagnosis

Demonstration of live adult lice, nymphs, or nits in pubic area to diagnose active infestation.

Course

Treatment is usually effective. Reinfestation can occur. Retreatment may be necessary if lice are found or if eggs are observed at hair–skin junction.

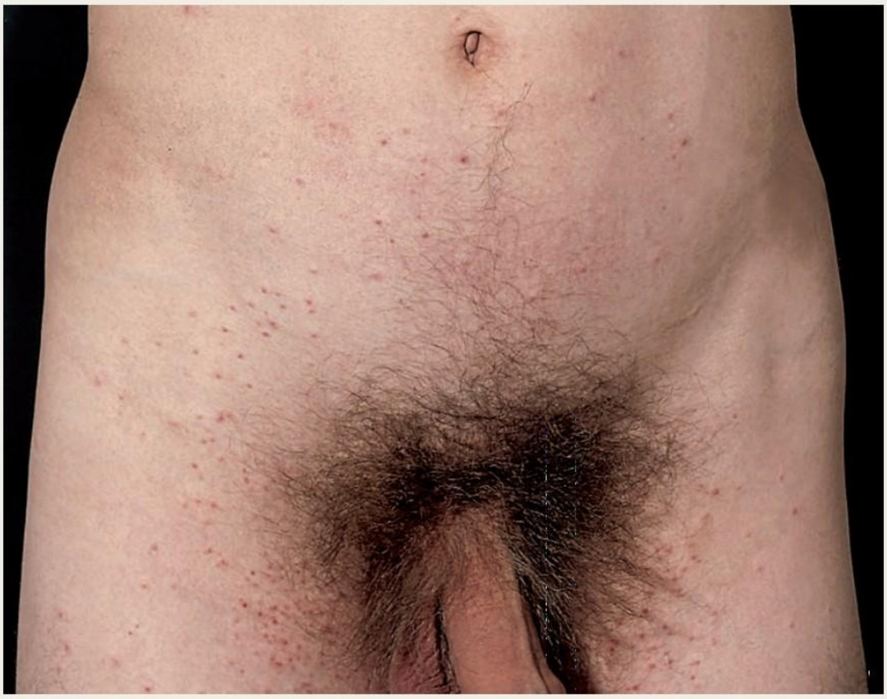

Figure 28-14. Crab lice infestation: papular urticaria A 25-year-old with pruritus. Multiple inflammatory papules at sites of crab lice bites on the abdomen and the inner aspects of the thighs.

Treatment

Pediculosis. See p. 705. Decontaminate bedding and clothing. Treat sex partners.

Demodicidosis ICD-9: 133.8 ∘ ICD-10: B88.0 ◨ ●

Demodex species are human face mites, part of the human cutaneous microbiome. *D. folliculorum* resides in hair follicles; *D. brevis*, infundibulum of sebaceous glands. Mites do not invade tissue. Site of habitation usually symptomatic. In some cases causes an inflammatory reaction (demodicidosis) that occurs with lesions resembling rosacea, suppurative folliculitis, or perioral dermatitis (Fig. 28-15).

- **Treatment.** Topical metronidazole, permethrin; in severe cases oral ivermectin 200 mg/kg.

Figure 28-16. Burrow with Sarcoptes scabiei (female), eggs, and feces Female mite at the end of a burrow with seven eggs and smaller fecal particles obtained from a papule on the webspace of the hand.

Clinical Manifestation

Symptoms

Patients are often aware of similar symptoms in family members or sexual partners. *Pruritus* is intense, widespread, usually sparing head and neck. Itching often interferes with or prevents sleep. Pruritus may be absent with hyperinfestation. *Rash* ranges from no rash to generalized erythroderma. Patients with atopic diathesis scratch, producing eczematous dermatitis. Some individuals experience pruritus for many months with no rash. Tenderness of lesions suggests secondary bacterial infection.

Cutaneous Findings

(1) Lesions occurring at the sites of mite infestation, (2) cutaneous manifestations of hypersensitivity to mites, (3) lesions secondary to chronic rubbing and scratching, (4) secondary infection, (5) hyperinfestation, and (6) variants of scabies in special hosts: those with an atopic diathesis, nodular scabies, scabies in infants/small children, scabies in the elderly.

Intraepidermal Burrows. Skin-colored ridges, 0.5–1 cm in length (Figs. 28-15, 28-17), either linear or serpiginous, with minute vesicle or papule at end of tunnel. Each infesting female mite produces one burrow. Mites are about 0.5 mm in length. Burrows average 5 mm in length but may be up to 10 cm. *Distribution*: Areas with few or no hair follicles, usually where stratum corneum is thin and soft, i.e., interdigital webs of hands, wrists, shaft of penis, elbows, feet, buttocks, axillae > (Fig. 28-18). In infants, infestation may occur on head and neck.

Scabies with nodules 5–20 mm in diameter, red, pink, tan, or brown in color, smooth (Fig. 28-19); burrow sometimes seen on the surface of a very early lesion. *Distribution*: Scrotum, penis, axillae, waist, buttocks, areolae (Fig. 28-20). Resolve with postinflammatory hyperpigmentation. May be more apparent after treatment, as eczematous eruption resolves.

Scabies with Hyperinfestation (formerly called Norwegian Scabies). May begin as ordinary scabies. In others, clinical appearance is of chronic eczema, psoriasiform dermatitis, seborrheic dermatitis, or erythroderma. Lesions often markedly hyperkeratotic and/or crusted (Figs. 28-21, 28-22). Warty dermatosis of hands/feet with nail bed hyperkeratosis. Erythematous scaling eruption on face,

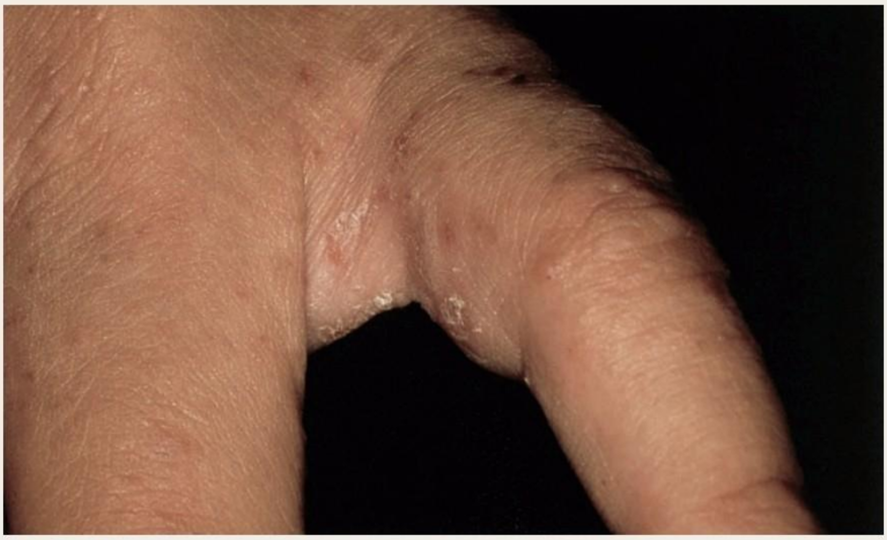

Figure 28-17. Scabies with burrows Papules and burrows in typical location on the finger web. Burrows are tan or skin-colored ridges with linear configuration with a minute vesicle or papule at the end of the burrow; they are often difficult to define.

deeply pigmented individuals. Bullous scabies roderma.

Laboratory Examinations

Microscopy. Highest yield in identifying a mite is in typical burrows on the finger webs, flexor aspects of wrists, and penis. A drop of mineral oil is placed over a burrow, and the burrow is scraped off with a curette or no. 15 scalpel blade and placed on a microscope slide. Three findings are diagnostic of scabies: *S. scabiei* mites, eggs, and fecal pellets (scybala) (Fig. 28-23).
Dermatopathology. *Scabietic burrow*: located within stratum corneum; female mite with eggs situated in blind end of burrow. Spongiosis (epidermal edema) near mite with vesicle formation common. Dermis shows infiltrate with eosinophils. Nodules: dense chronic inflammatory infiltrate with eosinophils. In some cases, persistent arthropod reaction resembling lymphoma with atypical mononuclear cells. Hyperinfestation: thickened stratum corneum riddled with innumerable mites.

Diagnosis

Clinical findings, confirmed, if possible, by microscopy (identification of mites, eggs, or mite feces).

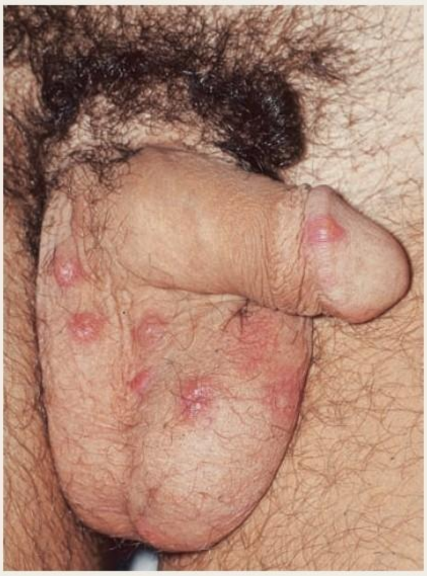

Figure 28-19. Scabies with nodules Red-brown papules and nodules on the penis and scrotum; these lesions are pathognomonic for scabies, occurring at sites of infestation in some individuals.

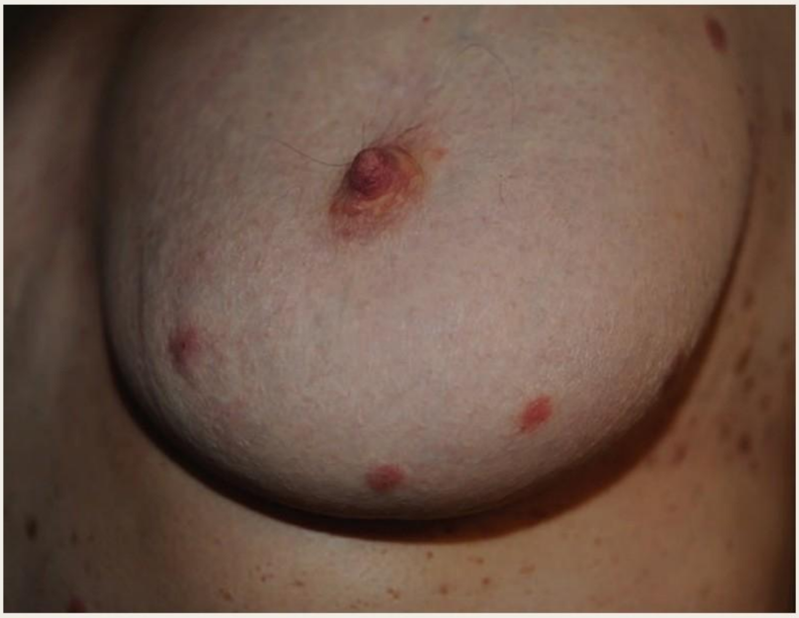

Figure 28-20. Scabies with nodules A 60-year-old female with reddish brown nodules on L-breast persisting after treatment with ivermectin.

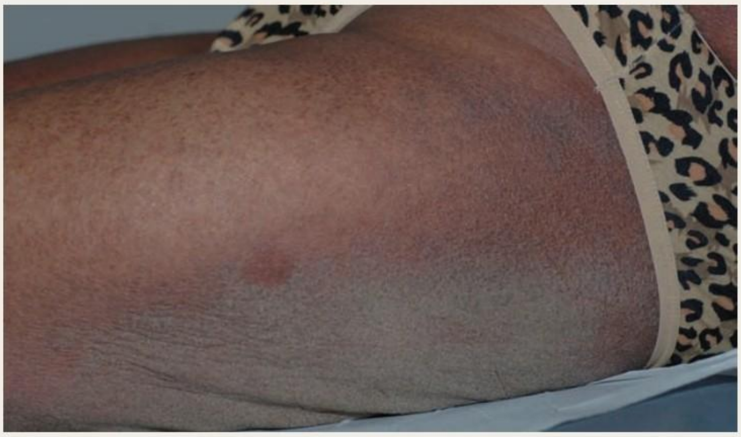

Figure 28-21. Scabies with hyperinfestation A 42-year-old Hispanic female with HTLV-I infection. Pruritus was minimal. Skin was hyperkeratotic and had a odor. Hundreds of burrows were seen on the back in Fig. 28-16.

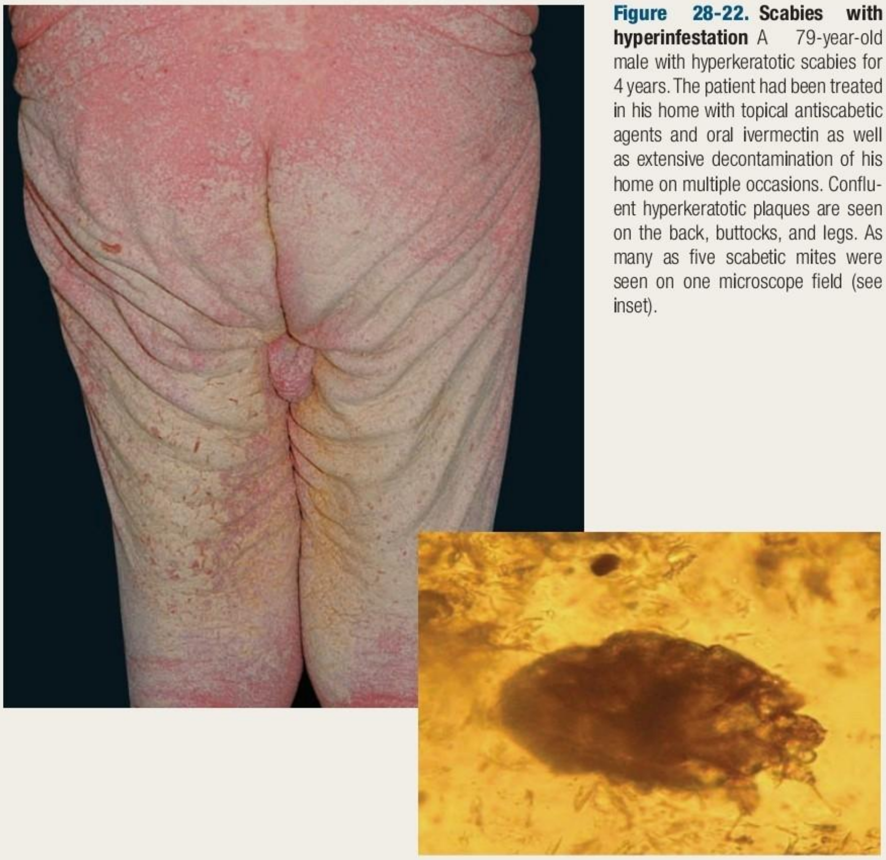

Figure 28-22. Scabies with hyperinfestation A 79-year-old male with hyperkeratotic scabies for 4 years. The patient had been treated in his home with topical antiscabetic agents and oral ivermectin as well as extensive decontamination of his home on multiple occasions. Confluent hyperkeratotic plaques are seen on the back, buttocks, and legs. As many as five scabetic mites were seen on one microscope field (see inset).

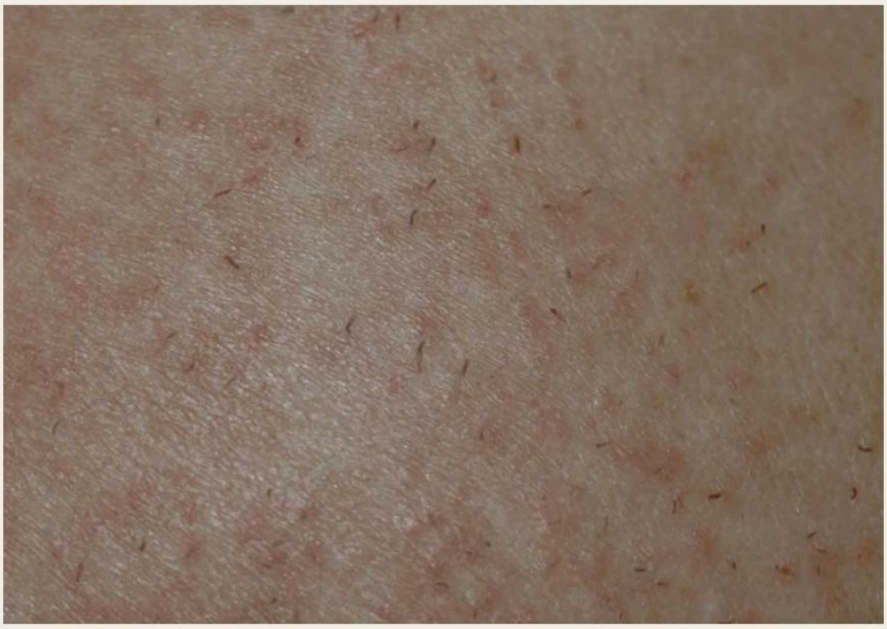

Figure 28-23. Scabies with multiple burrows A 42-year-old woman with HTLV-I infection and scabies with hyperinfestation (see Fig. 28-22). Multiple dark linear lesions on the back. Each of these lesions in an intraepidermal burrow created by a scabetic mite.

Course

Pruritus often persists up to several weeks after successful eradication of mite infestation, understandable in that the pruritus is a hypersensitivity phenomenon to mite antigen(s). If reinfestation occurs, pruritus becomes symptomatic within a few days. Delusions of parasitosis can occur in individuals who have been successfully treated for scabies or have never had scabies. Hyperinfestation: May be impossible to eradicate; recurrence more likely to relapse than reinfestation. Nodules: In treated patients, 80% resolve in 3 months but may persist up to 1 year.

Management

Principles of Treatment. Treat infested individuals and close physical contacts (including sexual partners) at the same time, whether or not symptoms are present. Application should be to all skin sites.

Recommended Regimens. Permethrin 5% Cream applied to all areas of the body. Lindane (g-Benzene Hexachloride) 1% lotion or cream applied thinly to all areas of the body from the neck down; wash off thoroughly after 8 h. Note: Lindane should not be used after a bath or shower, or by patients with extensive dermatitis, pregnant or lactating women, or children younger than 2 years. Mite resistance to lindane exists. Low cost makes lindane a key alternative in many countries.

Alternative Regimens. *Topical.* Crotamiton 10%, sulfur 2–10% in petrolatum, benzyl benzoate 10% and 25%, benzyl benzoate with sulfiram, malathion 0.5%, sulfram 25%, ivermectin 0.8%.

Systemic. Oral ivermectin, 200 μg/kg; single dose reported very effective in 15–30 days. Two to three doses, separated by 1–2 weeks, usually required for heavy infestation or in immunocompromised individuals. May effectively eradicate epidemic or endemic scabies in institutions such as nursing homes, hospitals, and refugee camps. Not approved by U.S. Food and Drug Administration or European Drug Agency. Do not use in infants, young children or pregnant/lactating women.

vans caused by *L. tropica*.

Pathogenesis

The clinical and immunologic spectrum of leishmaniasis parallels that of leprosy. CL occurs in a host with good protective immunity. MCL occurs in those with an intense inflammatory reaction. DCL occurs with extensive and widespread proliferation of the organism in the skin but without much inflammation or tendency for visceralization. VL occurs in the host with little immune response and/or in immunosuppression. Unlike leprosy, extent and pattern are strongly influenced by the specific species of *Leishmania* involved. Additional factors that affect the clinical picture: number of parasites inoculated, site of inoculation, nutritional status of host, and nature of the last nonblood meal of vector. Infection and recovery are followed by lifelong immunity to reinfection by the same species of *Leishmania*. In some cases, interspecies immunity occurs.

Incubation Period. Inversely proportional to size of inoculum: shorter in visitors to endemic area. OWCL: *L. tropica major*, 1–4 weeks; *L. tropica*, 2–8 months; acute CL: 2–8 weeks or more.

Symptoms. Noduloulcerative lesions usually asymptomatic. With secondary bacterial infection, may become painful.

NWCL: *L. mexicana* complex. Small erythematous papule develops at sandfly bite site, evolving into ulcerated nodule (Fig. 29-1). Enlarges to 3–12 cm with raised border. Nonulcerating nodules may become verrucous. Lymphangitis, regional lymphadenopathy. Isolated lesions on hand or head usually do not ulcerate. Eventually lesion heals with a depressed scar. Ear lesions may persist for years, destroying cartilage (chiclero ulcers) (Fig. 29-2).

ML. Characterized by naso-oropharyngeal mucosal involvement, a metastatic complication of CL. Mucosal disease usually becomes evident several years after healing of original cutaneous lesions; cutaneous and mucosal lesions can coexist or appear decades apart. Edema and inflammatory changes lead to epistaxis and coryzal symptoms.

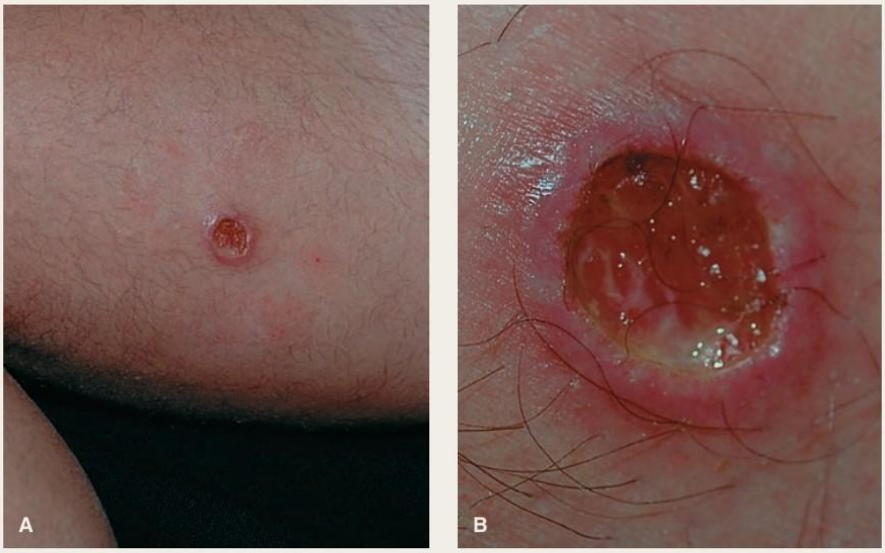

Figure 29-1. New World cutaneous leishmaniasis: ulcer on thigh A 42-year-old with HIV disease noted a painless lesion on the medial thigh 6 weeks after returning from Mexico **(A)** ulcer with rolled borders and base with granulation tissue **(B)**. Leishmania were seen on lesional biopsy. *L. mexicana* was isolated on tissue culture of lesional biopsy.

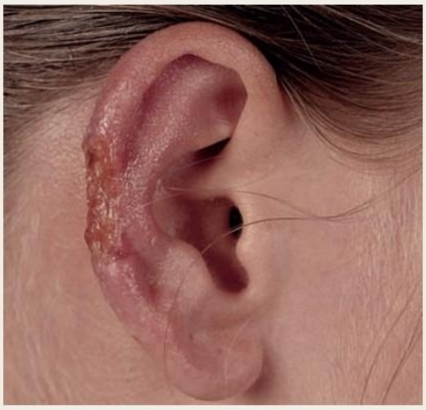

Figure 29-2. New World cutaneous leishmaniasis: chiclero ulcer A deep ulcer on the helix at the site of a sandfly bite. This variant typically occurs in leishmaniasis acquired in Central and South America.

In time, nasal septum, floor of mouth, and tonsillar areas destroyed (Fig. 29-3). Results in marked disfigurement (referred to as *espundia* in South America). Death may occur due to superimposed bacterial infection, pharyngeal obstruction, or malnutrition

OWCL. Begins as small erythematous papule, which may appear immediately after sandfly bite but usually 2–4 weeks later. Papule slowly enlarges to 2 cm over a period of several weeks and assumes a dusky violaceous hue (Figs. 29-4, 29-5). Eventually, lesion becomes crusted in center with a shallow ulcer and raised indurated border = volcano sign. In some cases, the center of the nodule becomes hyperkeratotic, forming a cutaneous horn. Small satellite papules may develop at periphery of lesion, and occasionally subcutaneous nodules along the course of proximal lymphatics. Peripheral extension usually stops after 2 months, and ulcerated nodule persists for another 3–6 months, or longer. The lesion then heals with a slightly depressed scar. In some cases, CL remains active with positive smears for 24 months (nonhealing chronic CL). The number of lesions depends on the circumstances of the exposure and extent of infection within the sandfly vector. May result in multiple lesions, up to 100 or more (Figs. 29-4, 29-5).

DCL. Resembles lepromatous leprosy; large number of parasites in macrophages in dermis; no visceral involvement. In Old World, occurs in 20% of individuals with leishmaniasis in Ethiopia and Sudan. In South America, attributed to a member of *L. braziliensis*

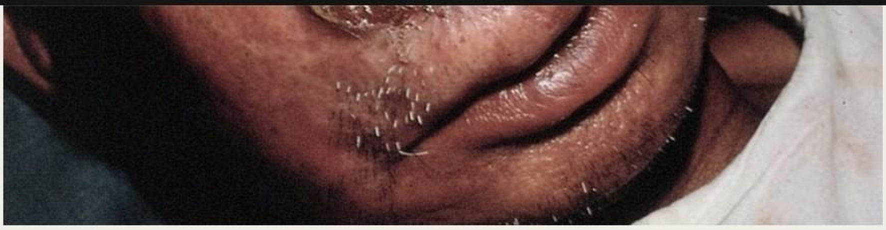

Figure 29-3. Mucocutaneous leishmaniasis: espundia Painful, mutilating ulceration with destruction of portions of the nose. (Courtesy of Eric Kraus, MD.)

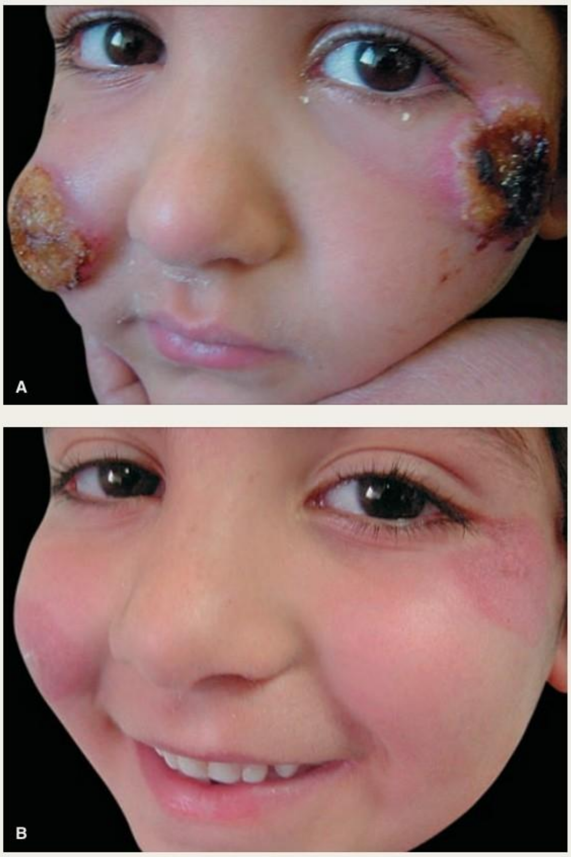

Figure 29-4. Old World cutaneous leishmaniasis: face A 7-year-old Jordanian girl with painful lesions on the cheeks for 6 weeks. **(A)** Large crusted nodules with surrounding edema on both cheeks. **(B)** Three weeks after successful therapy (sodium stibogluconate pentostam injections; 15 mg/kg per day IM injection for 21 days), lesions have healed with minimal residual erythema and no scarring. (Courtesy of Mohammad Tawara, MD.)

complex. Presents as a single nodule, which then spreads locally, often through extension from satellite lesions, and eventually by metastasis. In time, lesions become widespread with nonulcerating nodules appearing diffusely over face, trunk. Responds poorly to treatment.

Leishmaniasis Recidivans (LR). Complication of *L. tropica* infection. Dusky-red plaques with active, spreading borders and healing centers, giving rise to gyrate and annular lesions. Most commonly affects face; can cause tissue destruction and severe deformity.

PKDL. Sequel to VL that has resolved spontaneously or during/after adequate treatment. Lesions appear ≥1 year after course of therapy with macular, papular, nodular lesions, and hypopigmented macules/plaques on face (Fig. 29-6), trunk, and extremities. Resembles lepromatous leprosy when lesions are numerous. Develops in 20% of Indian patients treated for VL caused by *L. donovani* and in a small percentage of Ethiopian patients with VL caused by *L. aethiopica.*

VL. Can remain subclinical or become symptomatic, with acute, subacute, chronic course. Inapparent VL cases outnumber clinically apparent cases. Malnutrition is risk factor for clinically apparent VL. Bone marrow, liver, spleen are involved. Term *kala-azar* (Hindi for "black fever," some patients had gray color) refers to profoundly cachectic febrile patients with life-threatening disease. Patients present with fever, splenomegaly, pancytopenia, and wasting.

Differential Diagnosis

Acute CL. Insect bite reaction, impetigo, ecthyma, furuncle, *Mycobacterium marinum* infection, furuncular myiasis, chancre.

neously or during/after adequate treatment. Lesions appear ≥1 year after course of therapy with macular, papular, nodular lesions, and hypopigmented macules/plaques on face (Fig. 29-6), trunk, and extremities. Resembles lepromatous leprosy when lesions are numerous.

Differential Diagnosis

Acute CL. Insect bite reaction, impetigo, ecthyma, furuncle, *Mycobacterium marinum* infection, furuncular myiasis, chancre.

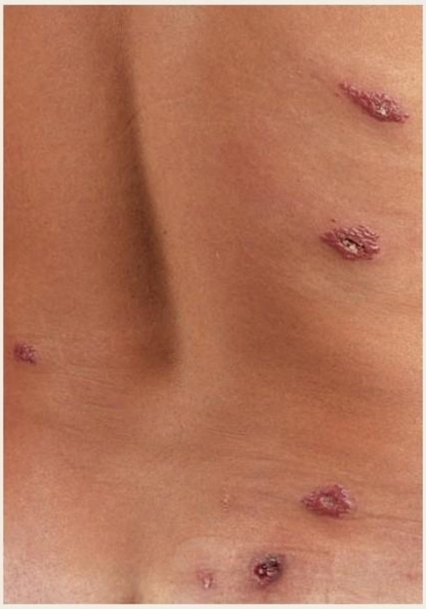

Figure 29-5. Old World cutaneous leishmaniasis Multiple, crusted nodules on the exposed back, arising at sites of sandfly bites. Many of the lesions resemble a volcano with a central depressed center, i.e., volcano sign.

Diagnosis

Clinical suspicion, confirmed by demonstrating:

- Intracellular nonflagellated amastigote in biopsy of skin, mucosa, liver, lymph nodes or aspirate of spleen, bone marrow, lymph node.
- Flagellated promastigote in culture of tissues (requires up to 21 days).

Course

In general, NWCL tends to be more severe and progressive than OWCL.

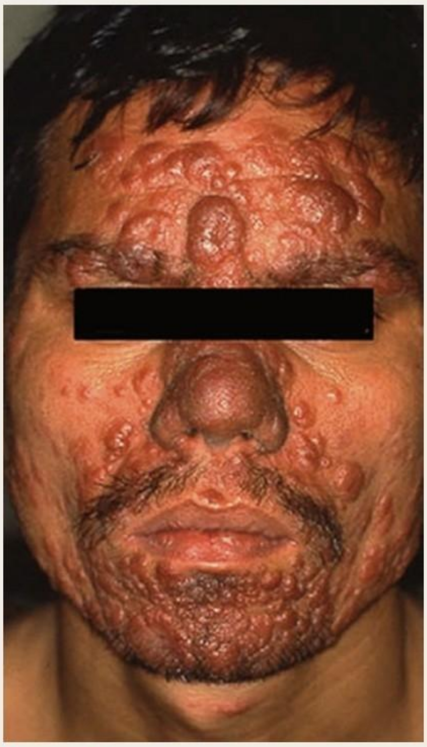
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Figure 29-6. Indian post-kala-azar dermal leishmaniasis. Coalescent erythematous dermal papules and nodules over the face in a picture similar to leonine facies. (Used with permission from Raj Kubba, MD.)

Treatment

Antimony-containing compounds meglumine antimoniate and sodium stibogluconate (Fig. 29-4) are given systemically. Other drugs used to treat leishmaniasis: amphotericin B, ketoconazole, miltefosine, paromomycin, and pentamidine.

Human American Trypanosomiasis

ICD-9: 086.9 ∘ ICD-10: B56 ICD-9: 086.0 ∘ ICD-10: B57

□ ◐ → ○

- *Synonym.* Chagas disease.
- **Etiology.** *Trypanosoma cruzi*
- **Demography.** Central and South America. 16–18 million persons infected.
- **Transmission.** *T. cruzi* deposited in feces of reduviid bugs onto the skin; enters host via breaks in skin (excoriations), mucous membranes, or conjunctivae. Can also be transmitted by transfusion of blood from infected persons, by organ transplantation, from mother to fetus.
- **Dissemination.** Via lymphatics and bloodstream to muscles.

Condyloma acuminatum. Cauliflower-floret (acuminate or pointed) lesions (Figs. 30-2 to 30-5).

Keratotic warts (Fig. 30-6).

Flat-topped papules/plaques (most common on cervix) (Fig. 30-7).

Skin-colored, pink, red, tan, brown. Solitary, scattered, and isolated, or form voluminous confluent masses. In immunocompromised individuals, lesions may be huge (Fig. 30-5).

Sites of predilection. Male: Frenulum, corona, glans penis, prepuce, shaft (Figs. 30-1, 30-2, 30-5, 30-6), scrotum. *Female*: Labia, clitoris, periurethral area, perineum, vagina, cervix (flat

larynx.
- Age: children <5 years of age, adults >20 years of age.
- Risk of SCCIS and invasive SCC.

Differential Diagnosis

Papular/Nodular External Genital Lesions. Normal anatomy (e.g., sebaceous glands, pearly penile papules, vestibular papillae), squamous intraepithelial lesions (SILs), SCCIS, invasive SCC, benign neoplasms (moles, seborrheic keratoses, skin tags, pilar cyst, angiokeratoma),

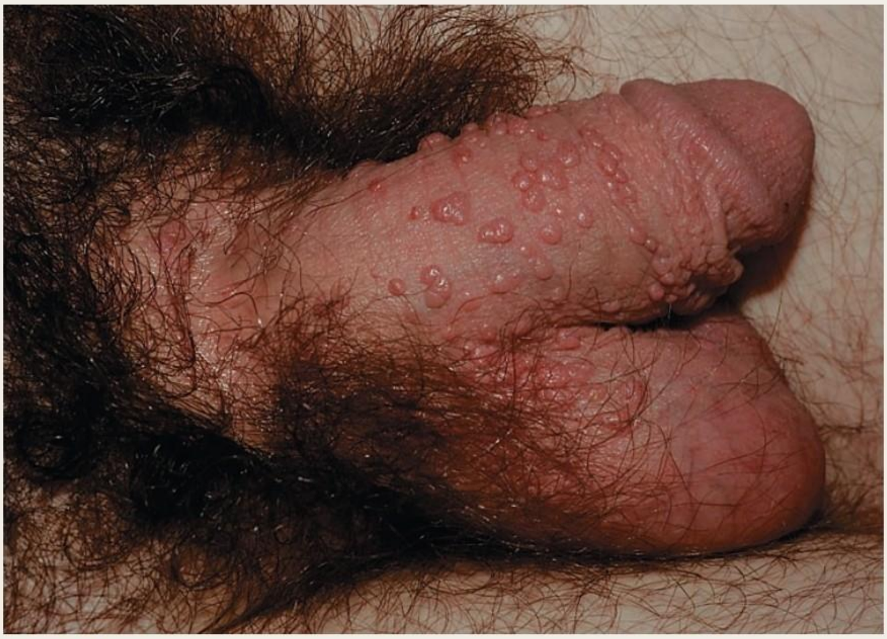


Figure 30-1. Papular warts: penis A 23-year-old male with penile lesions for 6 months. Multiple skin-colored papules on the penis and scrotum.

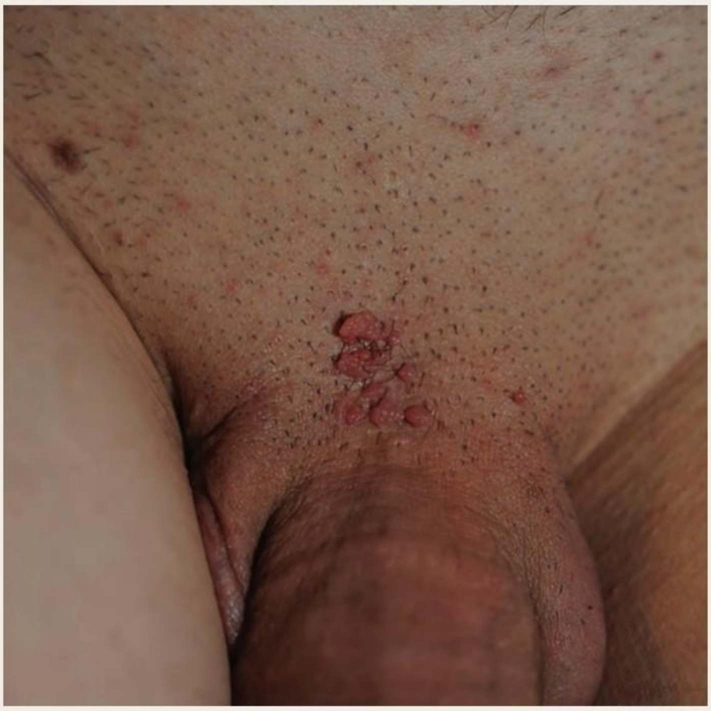


Figure 30-2. Condyloma acuminatum A 30-year-old male with cluster of warts at the base of the penis in pubis for 6 weeks. This is a common site for HPV infection; condom use does not protect against transmission from infected partner.

Figure 30-1. Papular warts: penis A 25-year-old male with penile lesions for 6 months. Multiple skin-colored papules on the penis and scrotum.

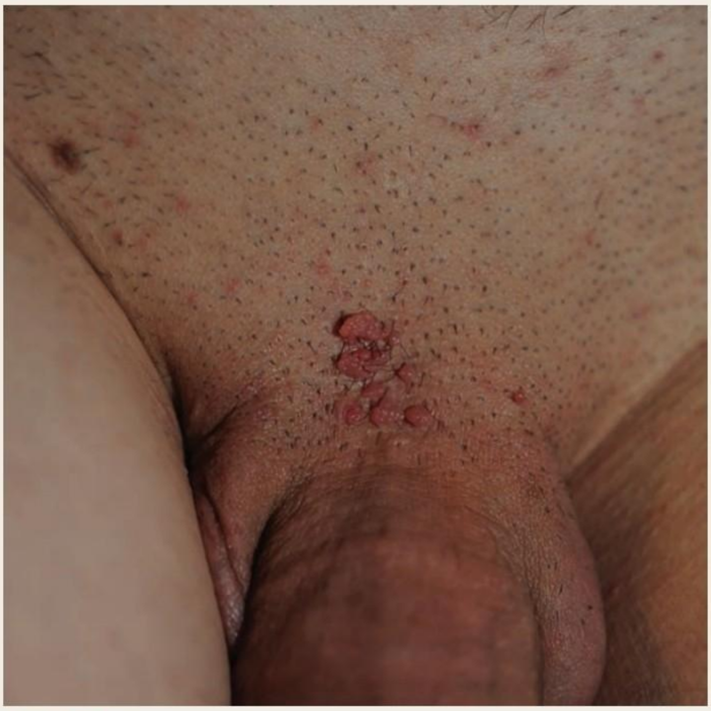


Figure 30-2. Condyloma acuminatum A 30-year-old male with cluster of warts at the base of the penis in pubis for 6 weeks. This is a common site for HPV infection; condom use does not protect against transmission from infected partner.

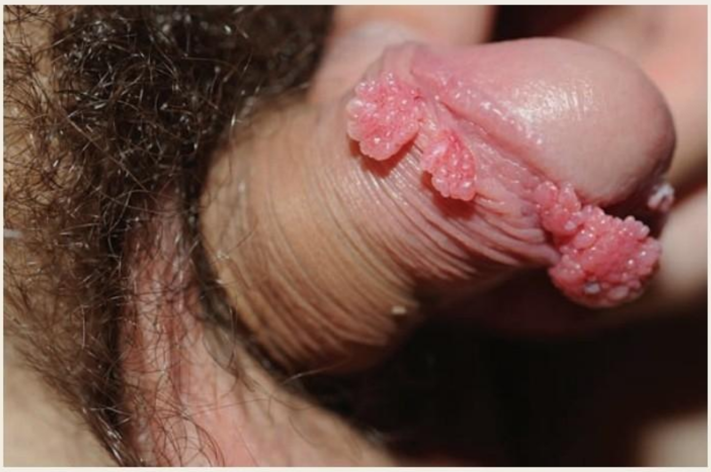


Figure 30-3. Condylomata acuminata: penis A 20-year-old male with Crohn disease treated with infliximab infusion. Condylomata on the distal foreskin resemble cauliflower floret-like papules.

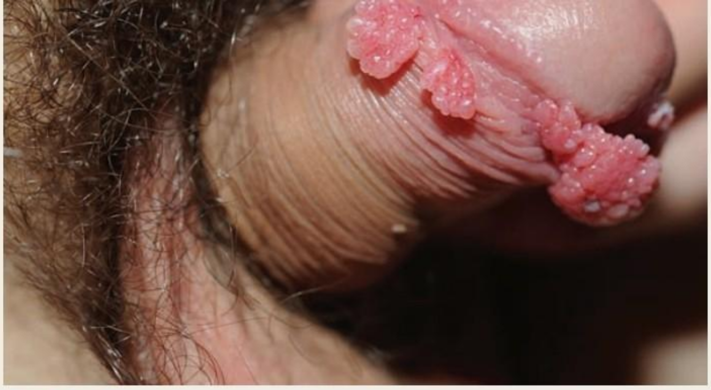


Figure 30-3. **Condylomata acuminata: penis** A 20-year-old male with Crohn disease treated with infliximab infusion. Condylomata on the distal foreskin resemble cauliflower floret-like papules.

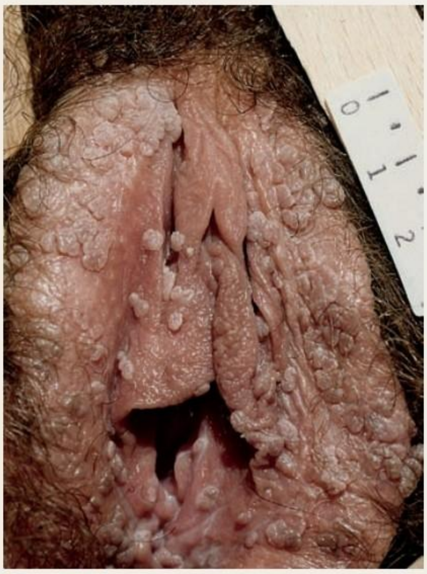


Figure 30-4. **Condylomata acuminata: vulva** Multiple, pink-brown, soft papules on the labia.

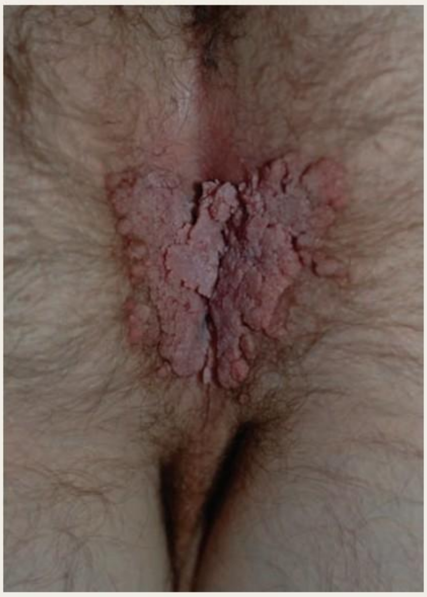


Figure 30-5. **Genital warts** A 37-year-old male with history of heart–lung transplantation and immunosuppression. Large condylomata acuminata are seen on the anal and perineal area.

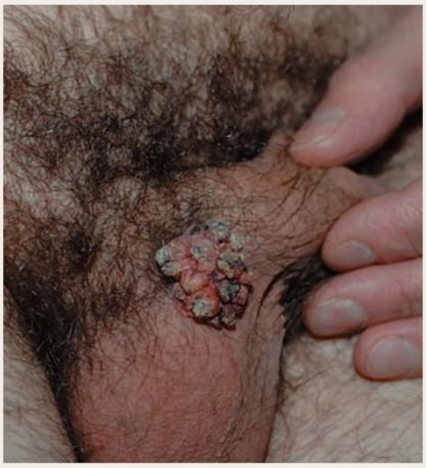


Figure 30-6. **Keratotic external genital warts (EGW): male** A 46-year-old male with lesion at the base of penis for several years. A keratotic tumor at the base of the penis adjacent to the scrotum. Lesional biopsy reported EGW ruling out verrucous carcinoma.

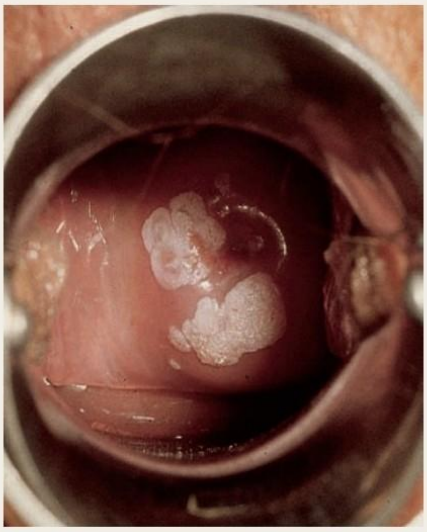


Figure 30-7. **Condylomata acuminata: uterine cervix** Sharply demarcated, whitish, flat plaques becoming confluent around the cervix.

Laboratory Studies

See Section 27 "Herpes Simplex Virus Disease."

Diagnosis

Diagnosis can be made on clinical finding. Confirmation by viral culture or direct fluorescent antibody (DFA) or serology may be indicated. Coinfection with another STD should be ruled out.

Course

GH is a lifetime infection and recurrances are the rule. Seventy percent are asymptomatic. Recurrence rates are high in those with an extended first episode of infection, regardless

Recurrances. Oral antivirals. Acyclovir 400 mg 3 times daily for 5 days or 800 mg twice daily for 5 days, or 800 mg 3 times daily for 2 days. Valacyclovir 500 mg twice daily for three days or 1 mg twice daily for 3 days. Famciclovir 125 mg twice daily for 5 days or 1 g once a day for 5 days.

Maintenance Therapy. Oral antivirals: Daily suppressive therapy. Acyclovir 400 mg twice daily. Valcyclovir 500–1000 mg once daily. Famciclovier 250 mg once daily.

Severely Immunocompromised. IV acyclovir 5 mg/kg every 8h for 5–7 days or oral acyclovir 400 mg 5 times a day for 7–14 days.

Acyclovir Resistant. IV foscarnet 40 mg/kg every 8h for 14–21 days.

Neonates. see Section 27.

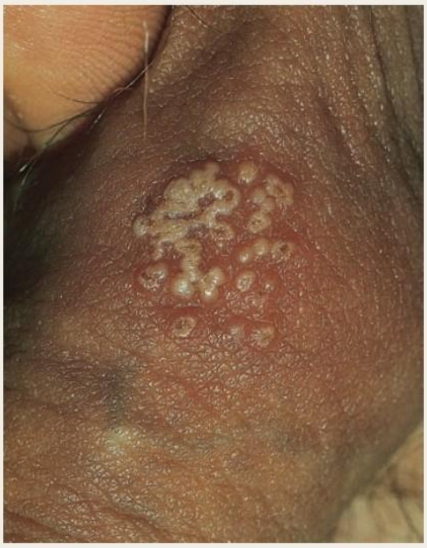


Figure 30-16. Genital herpes, recurrent Group of vesicles with early central crusting on a red base arising on the shaft of the penis. This "textbook" presentation, however, is much less common than small asymptomatic erosions or fissures.

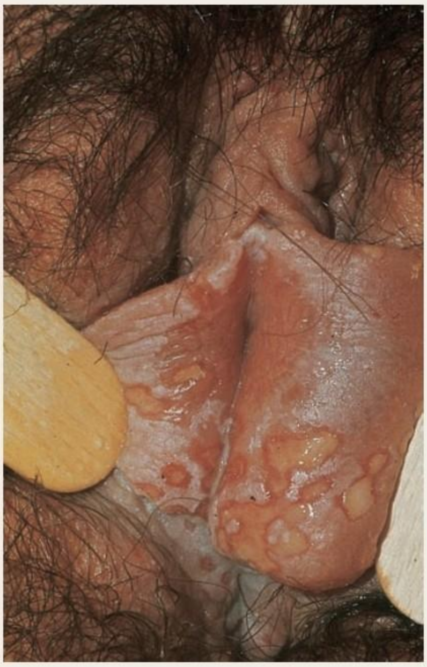


Figure 30-17. Genital herpes, recurrent: vulva Large, painful erosions on the labia. Extensive lesions such as these are uncommon in recurrent genital herpes in an otherwise healthy individual.

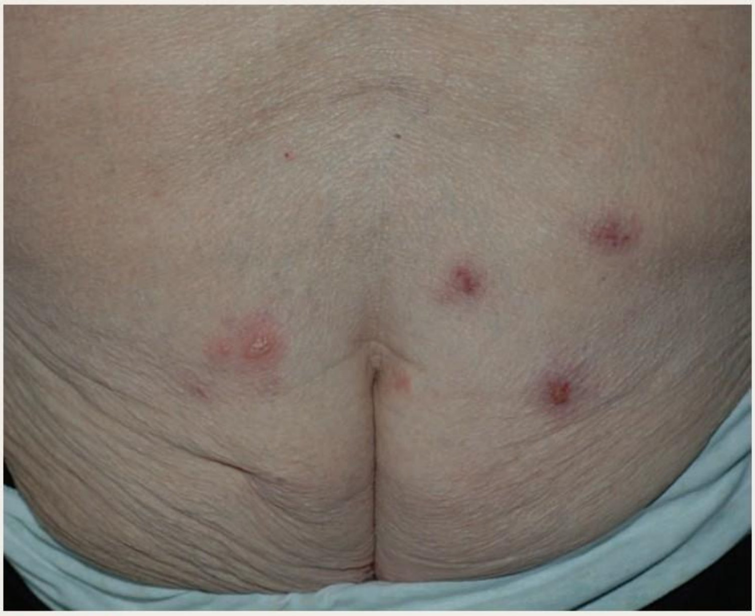


Figure 30-20. Genital herpes, recurrent A 80-year-old female with recurring lesions on buttock. She has polymyalgia rheumatic and is being treated with prednisone. Blisters and crusted erosions are seen on both buttocks.

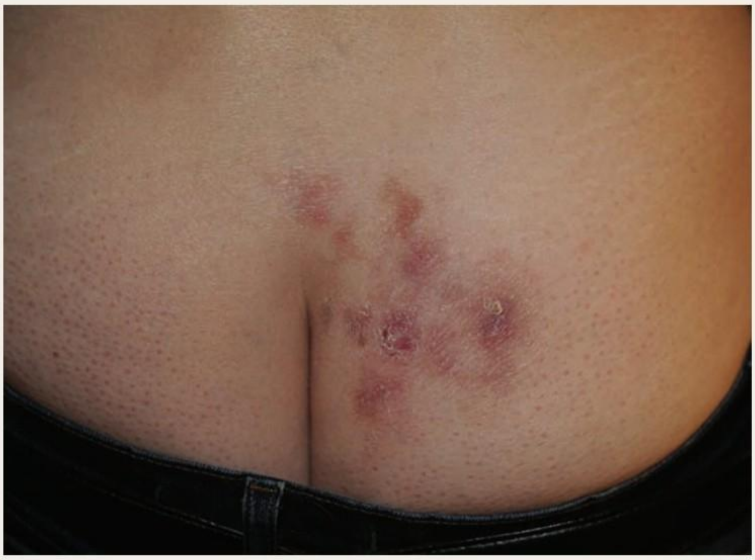


Figure 30-21. Genital herpes, recurrent A 51-year-old female with recurrent vesicles and crusted erosions on buttock, nearly continually since acquisition 31 years before. Recurrent lesion of the buttock were followed by erythema multiforme minor.

Neisseria Gonorrhoeae Disease

following therapy for early syphilis.

Treponemal STS FTA-ABS Test. Agglutination assays for antibodies to *T. pallidum*: Micro-

Treatment

Antibiotics (see p. 747–749 for specific doses). Educate patients and treat sex partners.

Primary Syphilis ICD-9: 91.2 ∘ ICD-10: A51 ☐ ◐

Clinical Manifestation

Genital or extragenital lesions occur at sites of inoculation. Ulcers are usually painless unless secondarily infected. Incubation period: 21 days (average); range, 10–90 days.

Chancre Button-like papule develops at the site of inoculation into a painless erosion and then ulcerates with raised border and scanty serous exudate (Figs. 30-25 to 30-27). Surface may be crusted. Lesions few millimeters to

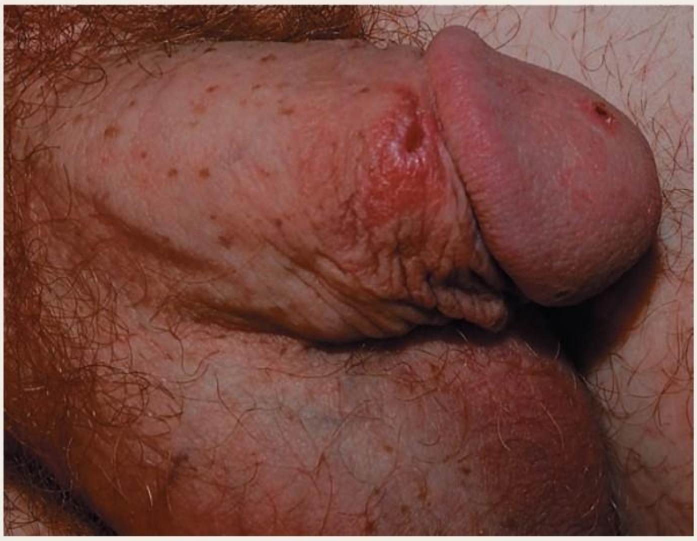


Figure 30-25. Primary syphilis: penile chancre A 28-year-old male with penile lesion for 7 days. Painless ulcer on distal penile shaft with smaller erosion on the glans. The ulcer is quite firm on palpation.

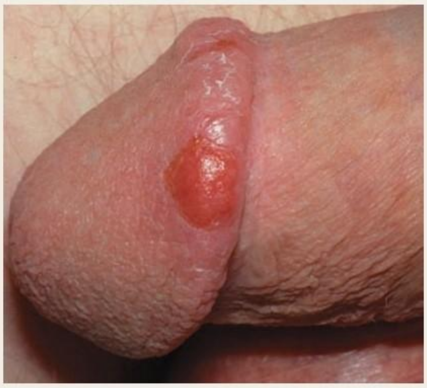


Figure 30-26. Primary syphilis: nodule on glans A 58-year-old male with penile lesion for 10 days. Red firm nodule on the glans; the lesion resolved without therapy and did not ulcerate. Biopsy reported inflammatory changes. The diagnosis was made in retrospect when STS obtained before marriage was positive.

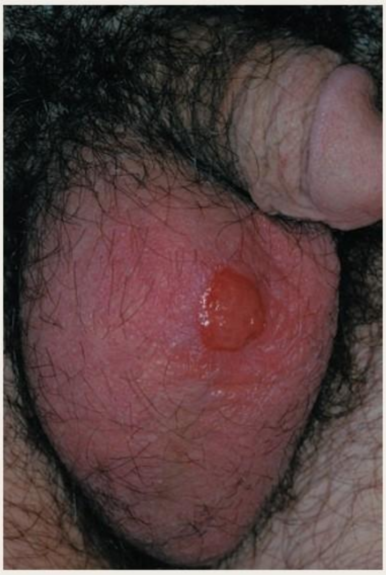


Figure 30-27. Primary syphilis: chancre on scrotum A 25-year-old male with painful lesion on scrotum for 10 days. A 1.5-cm ulcer on the scrotum, firm on palpation.

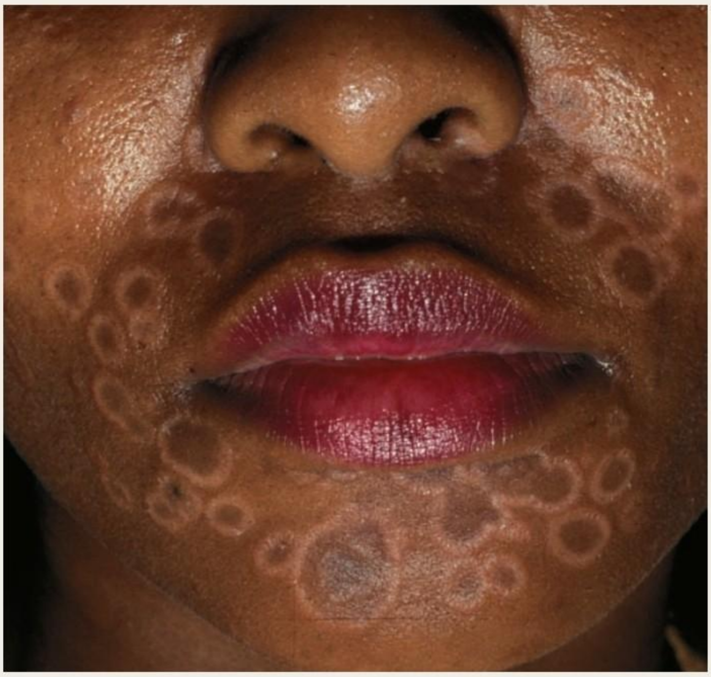


Figure 30-31. Secondary syphilis: annular facial lesions Annular plaques merging on the face of a South African woman. (Courtesy of Jeffrey S. Dover, MD.)

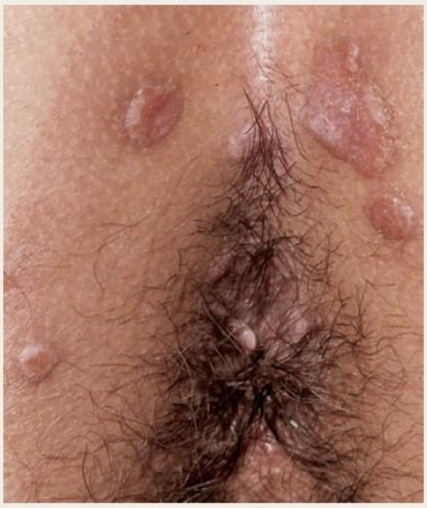


Figure 30-32. Secondary syphilis: condylomata lata Soft, flat-topped, moist, pink-tan papules and nodules on the perineum and perianal area. The lesions are teeming with *T. pallidum*.

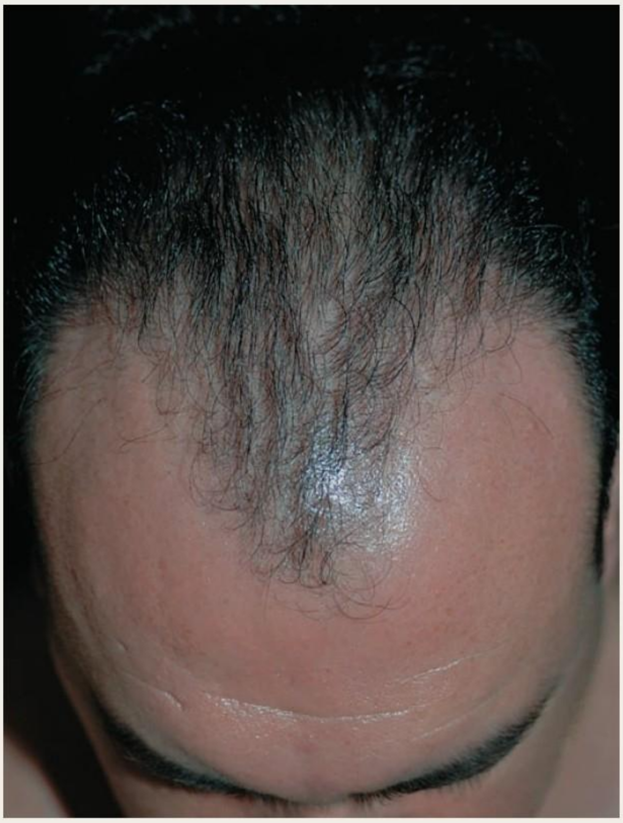


Figure 31-4. Pattern hair loss: male, Hamilton type III A 46-year-old male with bitemporal recession of hairline and frontal thinning of hair.

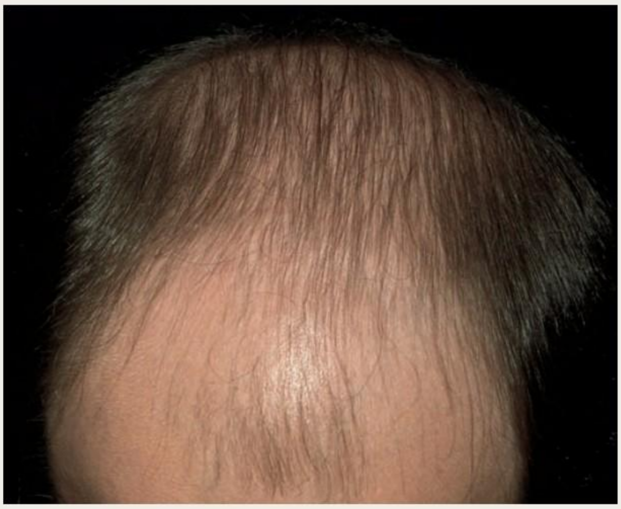
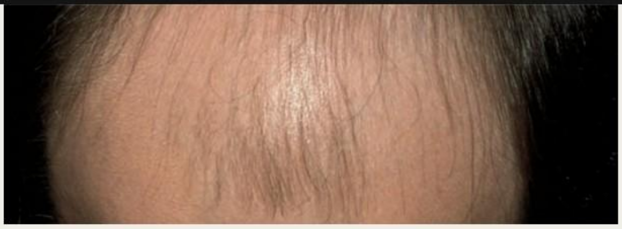


Figure 31-5. Pattern hair loss: male, Hamilton types IV to V A 37-year-old male with loss of hair in the frontotemporal and vertex areas in a male corresponding to Hamilton types IV and V.



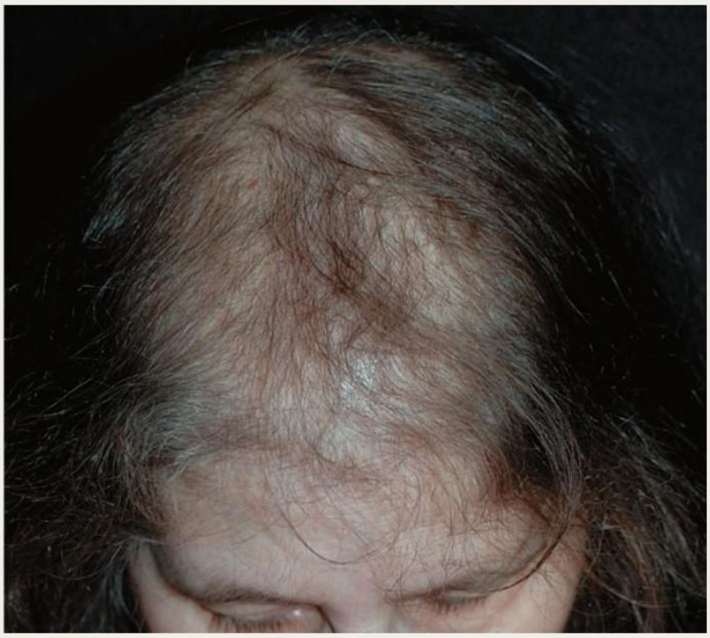


Figure 31-6. Pattern hair loss: female, Ludwig type II A 66-year-old female with diffuse thinning of hair on the crown.

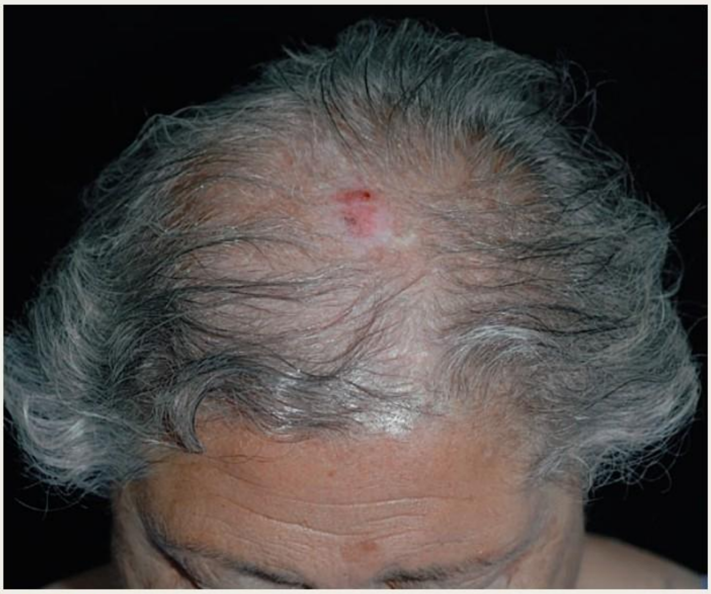


Figure 31-7. Pattern hair loss: female, Lugwig type III with basal cell carcinoma (BCC) A 67-year-old Greek female with advanced alopecia of the crown with BCC arising within it.

Hair

- Round patches of hair loss. Single or multiple. May coalesce. Alopecia often sharply defined

Laboratory Examinations

Serology. ANA (to rule out SLE); rapid plasma reagin (RPR) test (to rule out secondary syphilis).

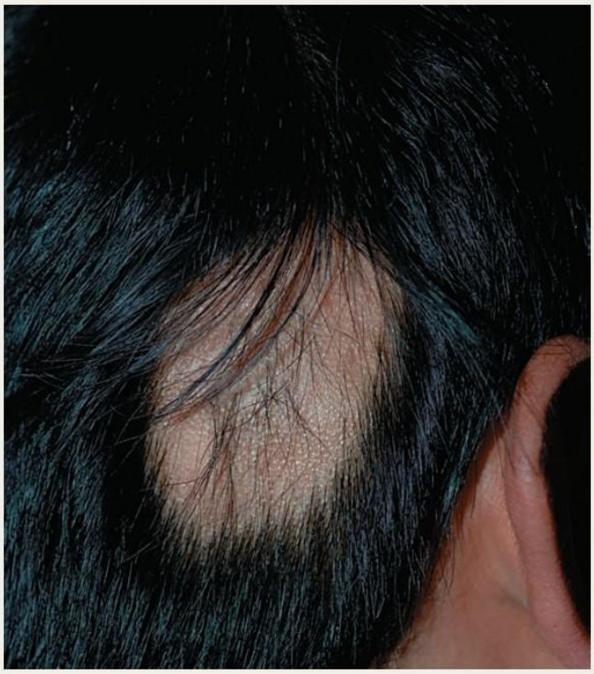


Figure 31-8. Alopecia areata (AA) of scalp: solitary lesion An area of alopecia without scaling, erythema, atrophy, or scarring on the occipital scalp. The short, broken-off hair shafts (so-called exclamation point hair) appear as very short stubs emerging from the bald scalp.

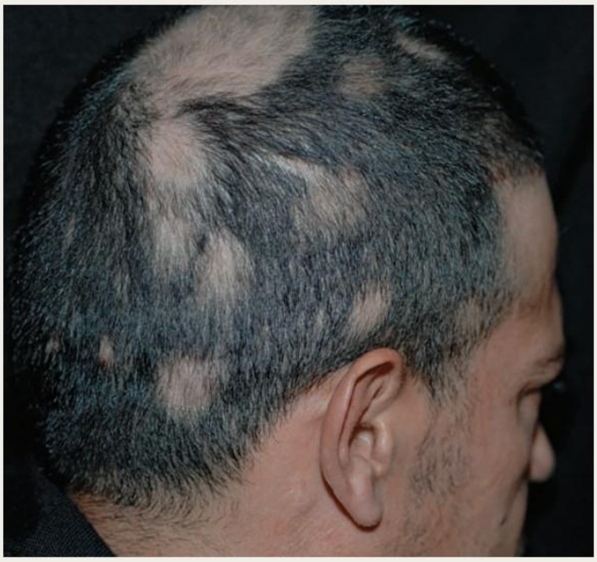


Figure 31-9. Alopecia areata of scalp: multiple, extensive lesions A 46-year-old male with multiple, confluent patches of alopecia areata.

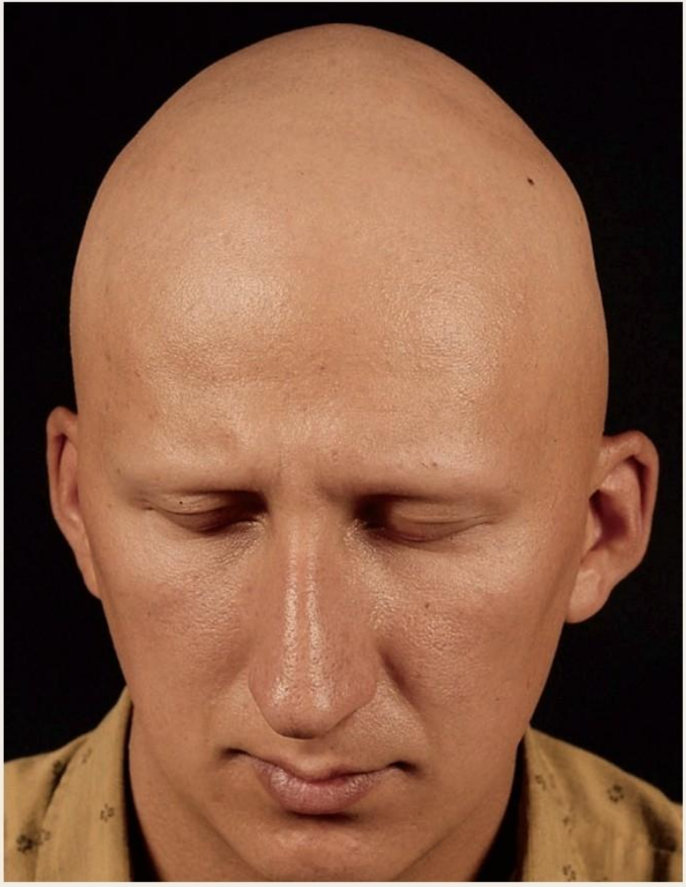


Figure 31-10. Alopecia areata universalis (AAU) This patient has lost all scalp hair (alopecia totalis), eyebrows, eyelashes, beard, and all body hair (alopecia universalis) and has dystrophic ("hammered brass") nails.

KOH Preparation. Rule out tinea capitis.

Dermatopathology. Acute lesions show peribulbar, perivascular, and outer root sheath mononuclear cell infiltrate of T cells and macrophages; follicular dystrophy with abnormal pigmentation and matrix degeneration. May show increased number of catagen/telogen follicles.

Course

- Spontaneous remission is common in patchy AA but is less so with AAT or AAU.
- Poor prognosis associated with onset in childhood, loss of body hair, nail involvement, atopy, family history of AA.
- If occurring after puberty, 80% regrow hair. With extensive involvement, <10% recover spontaneously.
- Recurrences of AA, however, are frequent.
- Systemic glucocorticoids or cyclosporine can induce remission of AA but do not alter the course.

Management

- Treatment directed at inflammatory infiltrate. No curative treatment is currently available.
- In many cases, the most important factor in management of the patient is psychological support from the dermatologist, family, and support groups (The National Alopecia Areata Foundation, http://-www.naaf.org/).

- Persons with extensive scalp involvement such as AAT may prefer to wear a wig or hairpiece.
- Makeup applied to eyebrows is helpful. Eyebrows can be tattooed.

Glucocorticoids. *Topical.* Superpotent agents not usually effective.

Intralesional Injection. Few and small lesions of AA can be treated with intralesional triamcinolone acetonide, 3–7 mg/mL, which can be very effective temporarily.

Systemic Glucocorticoids. May induce regrowth, but AA recurs on discontinuation; risks of long-term therapy therefore preclude their use.

Systemic Cyclosporine. Induces regrowth, but AA recurs when drug is discontinued.

Induction of Allergic Contact Dermatitis. Dinitrochlorobenzene, squaric acid dibutylester, or diphencyprone reported to be successful, but local discomfort due to allergic contact dermatitis and swelling of regional lymph nodes poses a problem.

Oral PUVA (Photochemotherapy). Variably effective, as high as 30%, and worth a trial in patients who are highly distressed about the problem. Entire body must be exposed.

Telogen Effluvium

☐ ●

- Telogen effluvium is the transient increased shedding of normal club (telogen) hairs from
- Results in increased daily hair loss and, if severe, diffuse thinning of scalp hair.

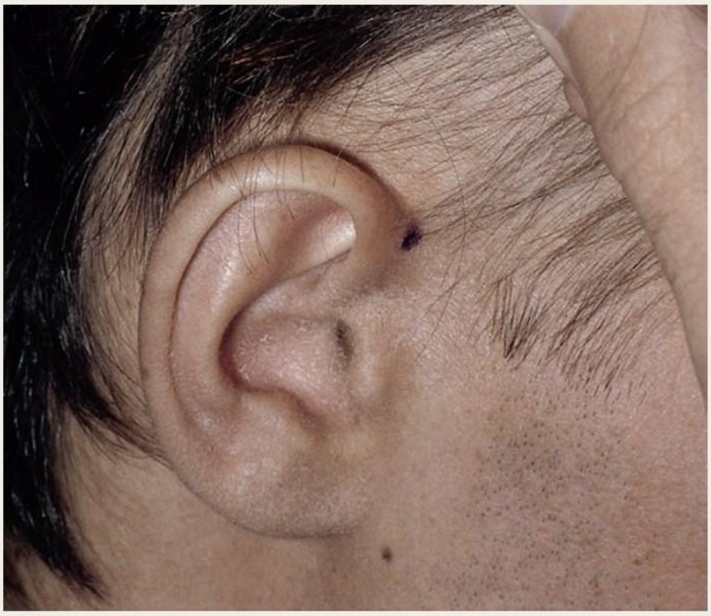


Figure 31-11. Telogen effluvium A clump of hair in the hand, associated with striking thinning of scalp hair. Using the fingers as shown, 30–40 hairs could be removed with each "hair pull."

Skin Lesions. No abnormalities of the scalp are detected.
Hair (Fig. 31-11). Diffuse shedding of the scalp hair. Gentle hair pull gathers several to many club or telogen hairs.
Distribution. Hair loss occurs diffusely throughout the scalp. Short regrowing new hairs are present close to the scalp; these hairs are finer than older hairs and have tapered ends.
Nails. The precipitating stimulus for TE may also affect the growth of nails, resulting in Beau lines (see Fig. 32-23), which appear as transverse lines or grooves on the fingernail and toenail plates.

Differential Diagnosis

Increased Shedding of Scalp Hair ± Nonscarring Alopecia. Pattern hair loss, diffuse-pattern alopecia areata, loose anagen syndrome, hyperthyroidism, hypothyroidism, SLE, secondary syphilis, drug-induced alopecia (Table 31-2).

Laboratory Examinations

Hair Pull. Compared with the normal hair pull, in which 80–90% of hair is in the anagen phase, TE is characterized by a reduced percentage of anagen hairs.
CBC. Rule out iron-deficiency anemia.
Chemistry. Serum iron, iron-binding capacity.
TSH. Rule out thyroid disease.
Serology. ANA, RPR.
Histopathology. Increase in the proportion of follicles in telogen.

Diagnosis

Made on history, clinical findings, hair pull, and possible biopsy, excluding other causes.

Course and Prognosis

Complete regrowth of hair is the rule. In postpartum TE, if hair loss is severe and recurs after successive pregnancies, regrowth may never be complete. TE may continue for up to a year after the precipitating cause.

Management

No intervention is needed or required. The patient should be reassured that the process is part of a normal cycle of hair growth.

Anagen Effluvium ☐ ◐

- Etiology: Radiation therapy to head; chemotherapy with alkylating agents; intoxications; protein malnutrition.
- Onset is usually rapid and extensive (see Fig. 31-12).
- More common and severe with combination chemotherapy than with the use of a single drug. Severity is generally dose dependent.
- Manifestations: *Scalp hair* loss is diffuse, extensive; also: eyebrows/lashes, beard, etc. *Nails*

- Etiology: Radiation therapy to head; chemotherapy with alkylating agents; intoxications; protein malnutrition.
- Onset is usually rapid and extensive (see Fig. 31-12).
- Pathogenesis: Occurs after any insult to hair follicle that impairs its mitotic/metabolic activity.
- More common and severe with combination chemotherapy than with the use of a single drug. Severity is generally dose dependent.
- Manifestations: *Scalp hair* loss is diffuse, extensive; also: eyebrows/lashes, beard, etc. *Nails* show transverse banding or ridging.
- Regrowth is usually rapid after discontinuation of chemotherapy.

Etiology

Anagen cycle disrupted causing varying degrees of hair follicle dystrophy:

- *Radiation therapy* to head.
- *Alkylating agents*: see Table 31-2.
- *Intoxications*: mercury, boric acid, thallium.
- Severe protein malnutrition.

Pathogenesis

- Occurs after any insult to hair follicle that impairs its mitotic/metabolic activity.
- Anagen hairs break off within the follicle or at the level of the scalp, being shed without roots.

Clinical Manifestations

Skin. Appears normal.

Hair. Scalp hair loss is diffuse, extensive (Fig. 31-12). Hair breaks off at the level of the scalp. Eyebrows/lashes, beard, body hair may also be lost.

Nails. Show transverse banding or ridging.

Course

- Hair regrows after discontinuation of chemotherapy.
- Regrowth after radiation depends on type, depth, dose fractionation; may result in irreversible hair follicle stem cell damage.

Management

No effective preventive measures are available.

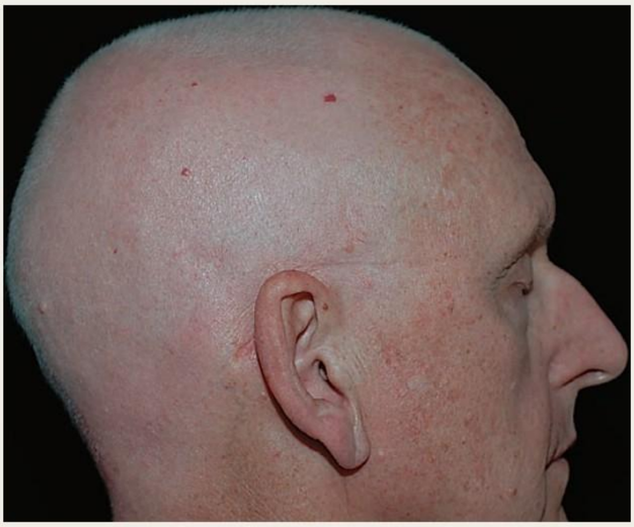


Figure 31-12. Anagen effluvium: chemotherapy All scalp, facial, and bodily hair have fallen out. Close inspection reveals that scalp hair has begun to regrow.

Cicatricial or Scarring Alopecia ■ ◐

- Primary cicatricial (scarring) alopecia results from damage or destruction of the hair follicles stem cells by:
    - Inflammatory (usually noninfectious) processes.
    - Infection: e.g., "kerion" tinea capitis, necrotizing herpes zoster.
    - Other pathologic processes: surgical scar, primary or metastatic neoplasm.
- Manifestations: Effacement of follicular orifices in a patchy or focal distribution, usually in scalp or beard.
- The end result is effacement of follicular orifices and replacement of the follicular structure by fibrous tissue (Table 31-3).
- Scarring is irreversible. Therapies are ineffective.

Chronic Cutaneous (Discoid) Lupus Erythematosus (CCLE): See Section 14.

- May occur without other manifestations or serologic evidence of lupus erythematosus.
- Manifestations:
    - CCLE: erythematous plaques (Figs. 31-13 to 31-15). Keratotic follicular plugs ("carpet tacks"). Scattered. Variable in number. May become confluent. Postinflammatory hypopigmentation, and/or follicular

- Follicular lichen planus (LP) is associated with cicatricial scalp alopecia, resulting in permanent hair loss (Fig. 31-16).
- LPP may or may not be associated with lichen planus of skin or mucosa.
- Most commonly affects middle-aged women.
- Manifestations in scalp: Perifollicular erythema ± hyperkeratosis. Violaceous discoloration of scalp. Prolonged inflammation results in scarring alopecia. In some cases, follicular

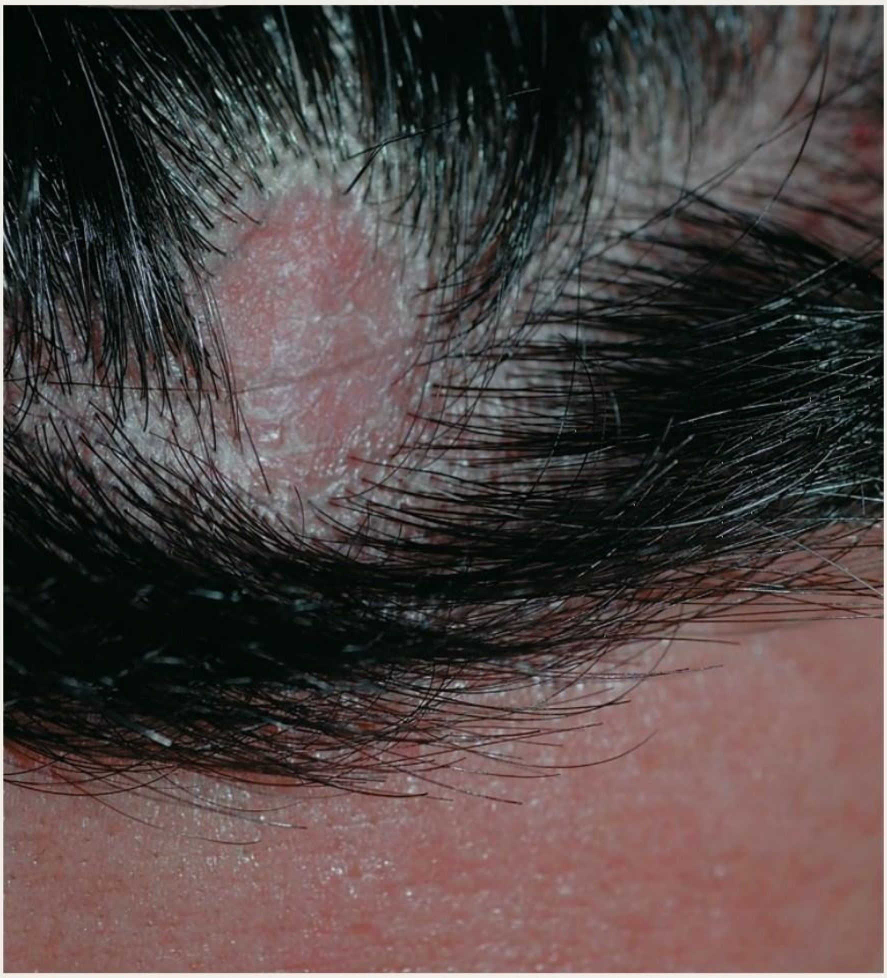


Figure 31-13. **Scarring alopecia of scalp: chronic cutaneous lupus erythematosus (CCLE)** A 41-year-old white male with multiple red discoid keratotic patches on the scalp for 1 year. A red scaling lesion with scarring alopecia is seen on the frontal scalp.

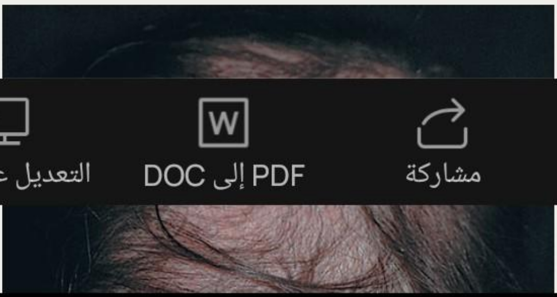


Figure 31-14. **Diffuse and scarring alopecia of scalp: Systemic LE (SLE) and CCLE lesions** A 36-year-old

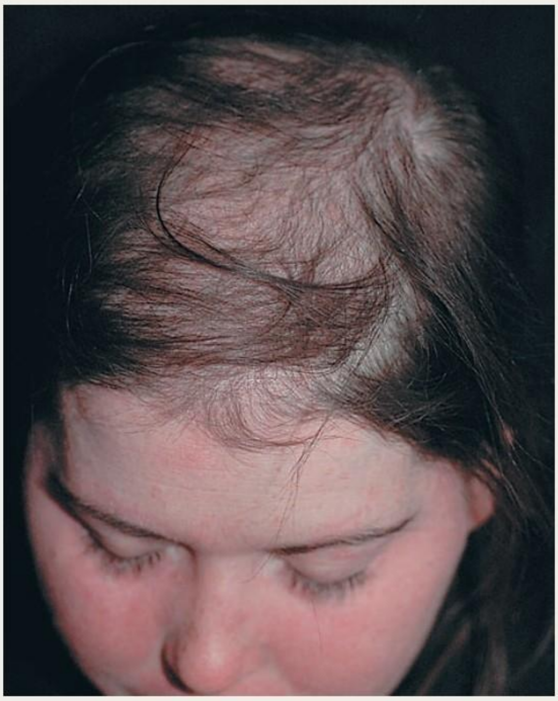


Figure 31-14. Diffuse and scarring alopecia of scalp: Systemic LE (SLE) and CCLE lesions A 36-year-old female with poorly controlled SLE for 3 years. Diffuse scalp alopecia is seen associated discrete discoid lesions with scarring alopecia.

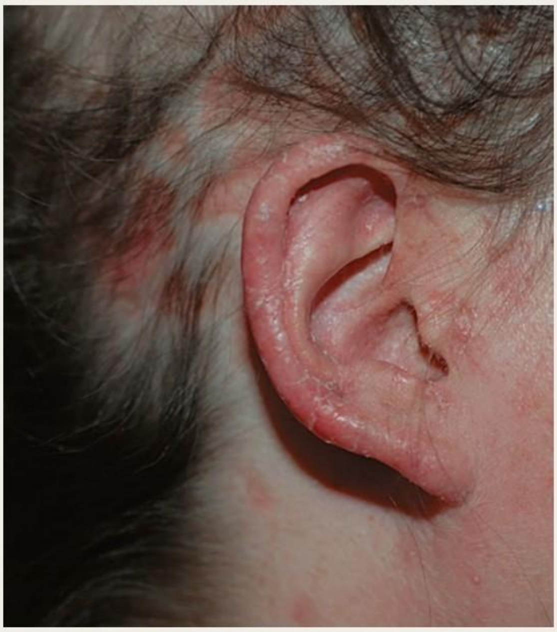
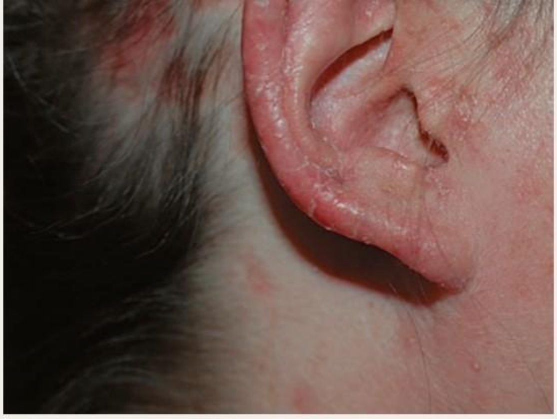


Figure 31-15. This is the same patient as in Fig. 31-14. She has erythema of the ears and red areas of scarring alopecia on the scalp.



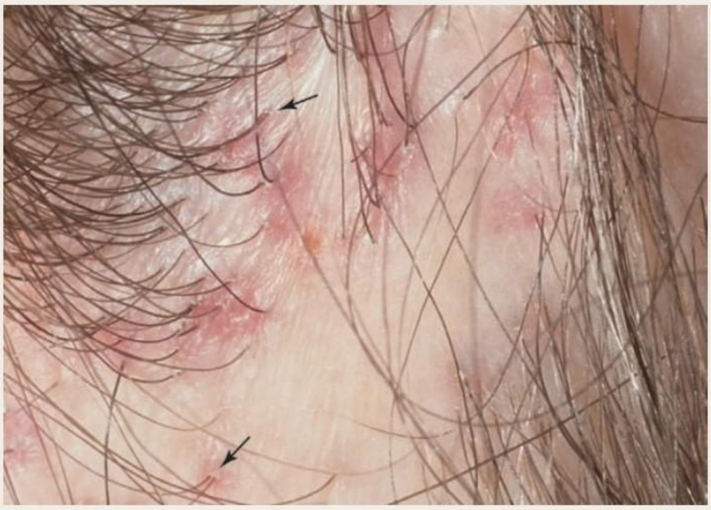


Figure 31-16. Scarring alopecia of scalp: pseudopelade of Brocq caused by lichen planus The scalp is smooth, shiny, devoid of hair and hair follicles in many areas; some of the remaining follicles are inflamed with perifollicular erythema and scale. Several hairs are seen emerging from a single site within the area of alopecia (*arrows*). The term pseudopelade implies that the lesions resemble alopecia areata.

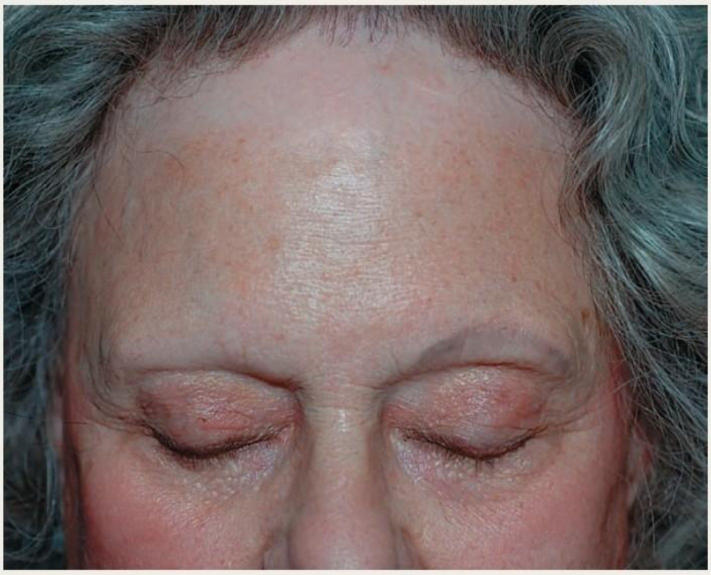


Figure 31-17. Scarring alopecia of scalp: lichen planopilaris (LPP) The frontal hairline has gradually receded; the area of alopecia lacks the pigmentation of forehead skin, which has had lifelong sun exposure. Both eyebrows have no hair; the eyebrow on the right is penciled in. The eyelashes appear normal. No other clinical findings of LP were detected. This clinical variant of LPP is called frontal fibrosing alopecia.

licular lymphohistiocytic infiltrate without concentric lamellar fibrosis.

- Initial deep inflammatory nodules, primarily over the occiput, that progress to coalescing regions of boggy scalp (Fig. 31-20). Sinus tracts may form; purulent exudates can be expressed. *S. aureus* secondary infection is common.

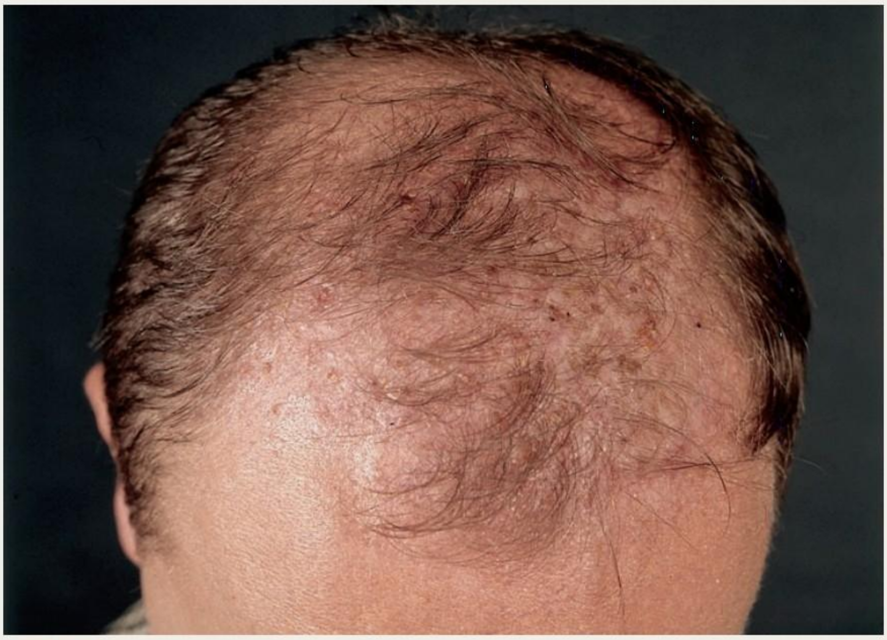


Figure 31-19. Scarring alopecia of scalp: folliculitis decalvans Erythema, inflammatory papules, crusts, and scarring of the scalp. Male pattern hair loss is also present.

- Dermatopathology: early follicular plugging and suppurative follicular/perifollicular abscesses with mixed inflammatory infiltrate; later, foreign-body giant cells, granulation tissue, scarring with sinus tracts.
- Scarring alopecia is irreversible. *S. aureus* infection should be documented and treated with appropriate antimicrobial agent.

Follicultis Keloidalis Nuchae

- *Synonym*: acne keloidalis (nuchae).
- Occurs most commonly in black men.
- Usually occurs on the occipital scalp and nape of the neck, starting with a chronic papular or pustular eruption (Fig. 31-21). Keloidal scar formation may occur.
- Distribution: nape of the neck, occipital scalp.
- Early mild involvement may respond to intralesional triamcinolone. If *S. aureus* is isolated on culture, treat with appropriate antimicrobial agent.

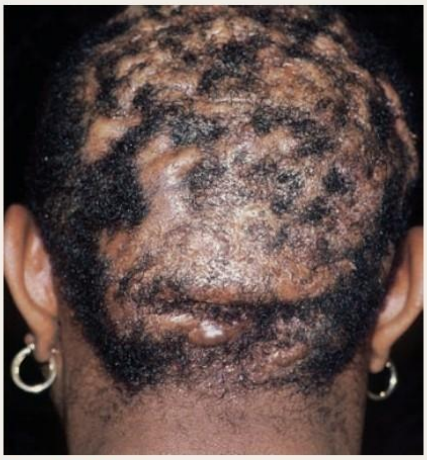


Figure 31-20. Scarring alopecia of scalp: dissecting folliculitis A 46-year-old black female with longstanding abscess formation of the scalp has resulted in very severe hypertrophic scarring. There was associated cystic acne and hidradenitis suppurativa.

Pseudofolliculitis Barbae

- *Synonym*: "razor bumps."
- Occurs commonly in black men who shave.
- Related to curved hair follicles. Cut hair retracts beneath skin surface, grows, and penetrates follicular wall (transfollicular type) or surrounding skin (extrafollicular

tion tissue, scarring with sinus tracts.
- Scarring alopecia is irreversible. *S. aureus* infection should be documented and treated with appropriate antimicrobial agent.

Follicultis Keloidalis Nuchae

- *Synonym*: acne keloidalis (nuchae).
- Occurs most commonly in black men.
- Usually occurs on the occipital scalp and nape of the neck, starting with a chronic papular or pustular eruption (Fig. 31-21). Keloidal scar formation may occur.
- Distribution: nape of the neck, occipital scalp.
- Early mild involvement may respond to intralesional triamcinolone. If *S. aureus* is isolated on culture, treat with appropriate antimicrobial agent.

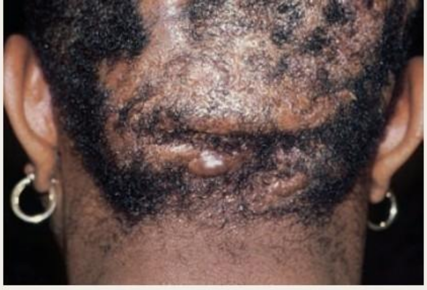


Figure 31-20. Scarring alopecia of scalp: dissecting folliculitis A 46-year-old black female with longstanding abscess formation of the scalp has resulted in very severe hypertrophic scarring. There was associated cystic acne and hidradenitis suppurativa.

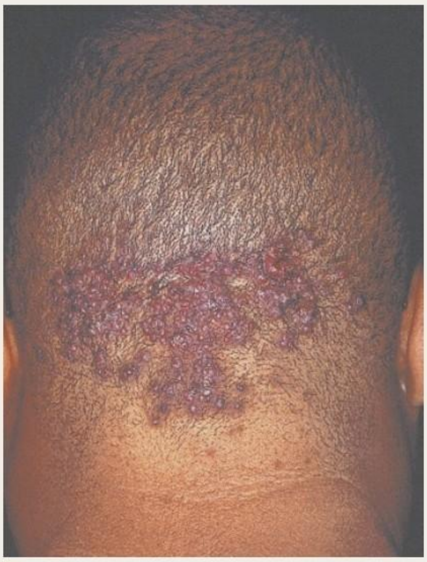


Figure 31-21. Scarring alopecia of scalp: folliculitis keloidalis A 31-year-old black male with papular scars of 3 years' duration, and follicular pustules becoming confluent on the occipital scalp and neck.

Pseudofolliculitis Barbae

- *Synonym*: "razor bumps."
- Occurs commonly in black men who shave.
- Related to curved hair follicles. Cut hair retracts beneath skin surface, grows, and penetrates follicular wall (transfollicular type) or surrounding skin (extrafollicular type), causing a foreign-body reaction.
- Distribution: any shaved area, i.e., beard (Fig. 31-22), scalp, pubic.
- Keloidal scarring in varying degrees occurs at involved sites.
- *S. aureus* secondary infection is common.

Acne Necrotica

- Pruritic or painful erythematous follicular-based papule with central necrosis, crusting, and healing with depressed scar.
- Lesions occur on anterior scalp, forehead, nose; at times, the trunk.
- Dermatopathology: lymphocytic necrotizing folliculitis.

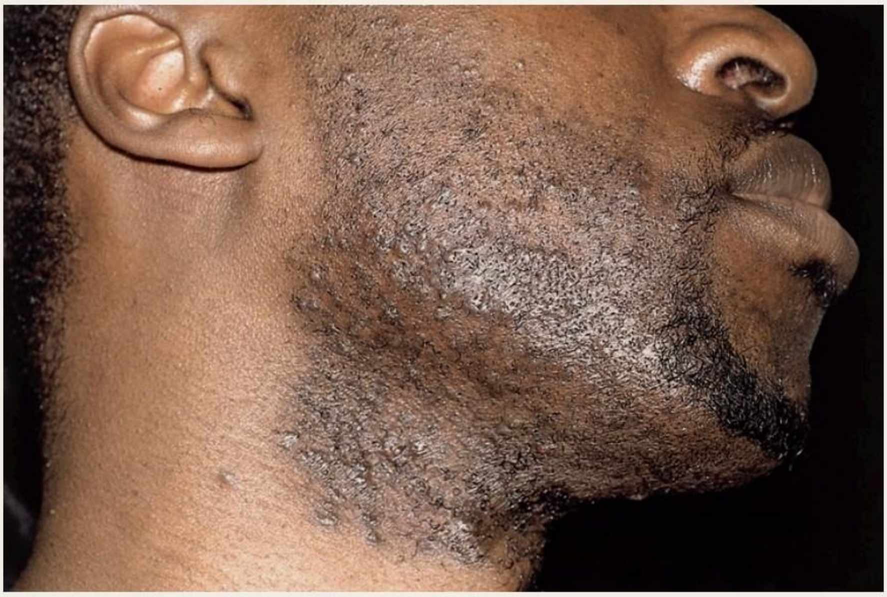


Figure 31-22. Pseudofolliculitis barbae A 29-year-old black male with multiple follicular papular scars in the beard; the presence of follicular pustules usually indicates secondary *Staphylococcus aureus* folliculitis. Folliculitis keloidalis is often seen on the occipital scalp and neck (see Fig. 31-21).

drome is suspected.

Laboratory Evaluation of Hirsutism

Serum Testosterone. If >200 ng/mL, exclude androgen-secreting tumor.

gens. Spironolactone (100–200 mg daily). Cyproterone acetate. Finasteride.

Oral Contraceptives. Inhibit androgen synthesis by inhibiting output of gonadotropins; most effective if combined with antiandrogens.

Bromocriptine. For treatment of prolactinoma.

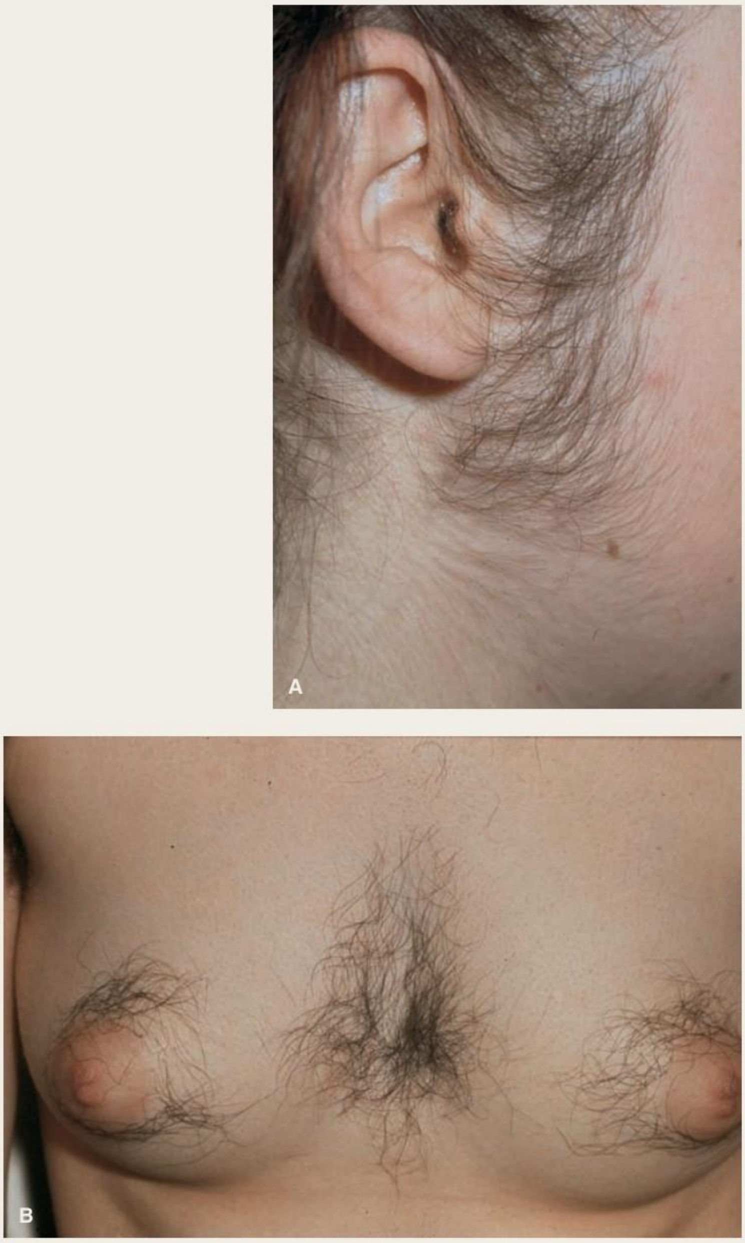


Figure 31-23. **Hirsutism: face and chest. (A)** Increased hair growth in androgen-dependent hair follicles of the sideburn area, associated with androgen excess. **(B)** Increased hair growth in androgen-dependent hair follicles of the presternal and periareolar regions.

Hypertrichosis ■ ◐

- Hypertrichosis is excessive hair growth (density, length) beyond accepted limits of normal for age, race, sex in areas that are not androgen sensitive
- May be generalized/universal or localized.
- May consist of lanugo, vellus, or terminal hair.

Hypertrichosis ■ ◐

- Hypertrichosis is excessive hair growth (density, length) beyond accepted limits of normal for age, race, sex in areas that are not androgen sensitive (see Fig. 31-24).
- May be generalized/universal or localized.
- May consist of lanugo, vellus, or terminal hair.

Etiology

Congenital or hereditary; acquired (see "Acquired Hypertrichosis Lanuginosa," below), drugs (minoxidil, phenytoin, cyclosporine, glucocorticoids, streptomycin, PUVA), porphyria, POEMS syndrome, hypothyroidism.

Clinical Manifestation

Localized Hypertrichosis. Trauma/scar/occupation-related sites of irritation. Drug-induced: topical minoxidil. Becker nevus.

Acquired Hypertrichosis Lanuginosa. Production of lanugo (wasp) hair in follicles previously producing vellus hair ("malignant down"). Hair may be >10 cm in length in nonscalp areas. Can involve entire body, except for palms and soles. In mild types, downy hair is limited to the face; hair on previously hairless areas such as the nose and eyelids is usually noticed first.

Universal Hypertrichosis (Fig. 31-24). Increase of lanugo, vellus, or terminal hair.

Management

- Find and remove the inciting cause.
- Similar to "Cosmetic Treatment" of hirsutism (see above).

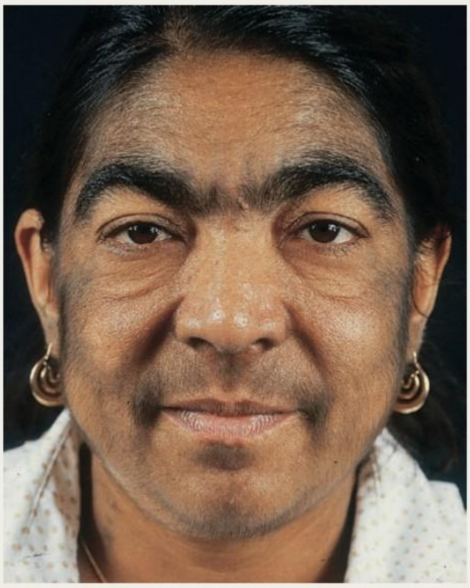


Figure 31-24. Hypertrichosis of face Excessive hair growth in nonandrogen-sensitive areas of the face in a female treated with cyclosporine.

Infectious Folliculitis ICD-9: 704.8 ∘ ICD-10: L73.8 ☐ ◐

- Infectious folliculitis begins in the upper portion of the hair follicle.
- Etiologic agents: Bacteria, fungi, virus, mites.
- Manifestations: Follicular papule, pustule, erosion, or crust at the follicular infundibulum.
- Infection can extend deeper into the entire length of the follicle (sycosis).

Etiology and Epidemiology

Etiology

Bacterial: *S. aureus* (Bockhart impetigo);

Skin Lesions

- Papule or pustule confined to the ostium of the hair follicle, at times surrounded by an

	fever who lie in supine position)
Buttocks	*S. aureus*, dermatophytes

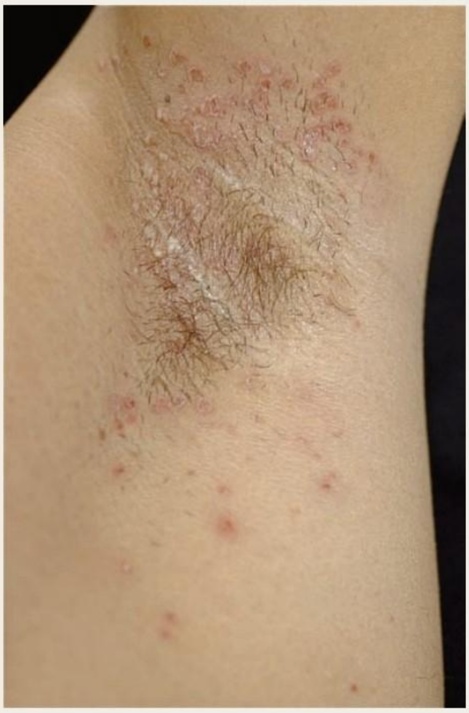


Figure 31-25. Infectious folliculitis, superficial in axilla: MRSA A 25-year-old male with pruritic and tender axillary lesions for several weeks. Multiple follicular pustules and papules are seen in the vault of the shaved axilla. Shaving facilitates entry of *S. aureus* into the superficial hair follicle. The lesions resolved with minocycline.

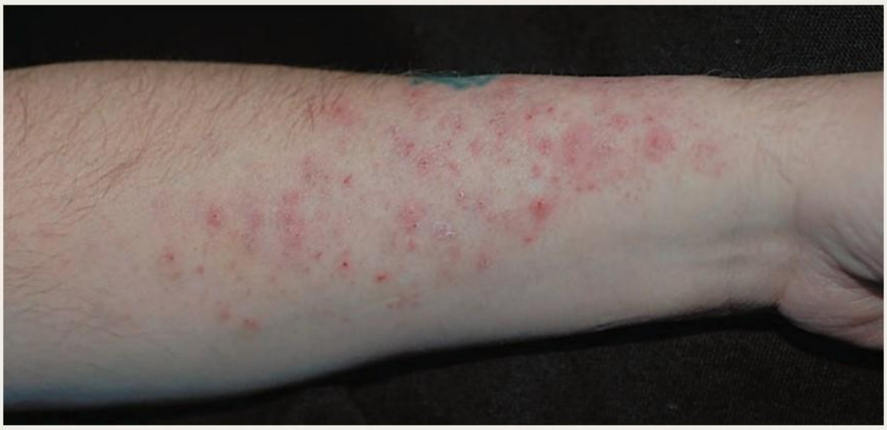
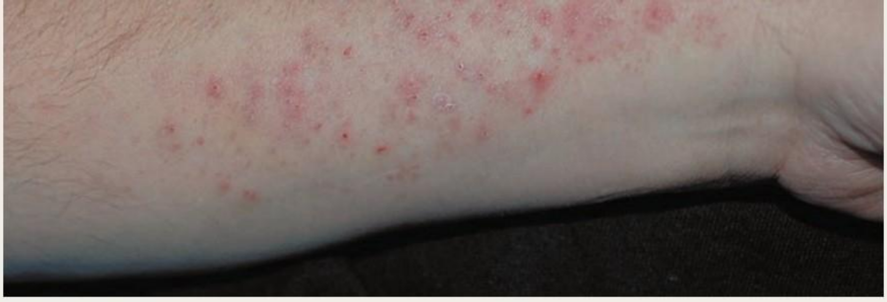


Figure 31-26. Infectious folliculitis on forearm A 44-year-old male with HIV/AIDS and numerous pustules and papules with superimposed mild lichen simplex chronicus.



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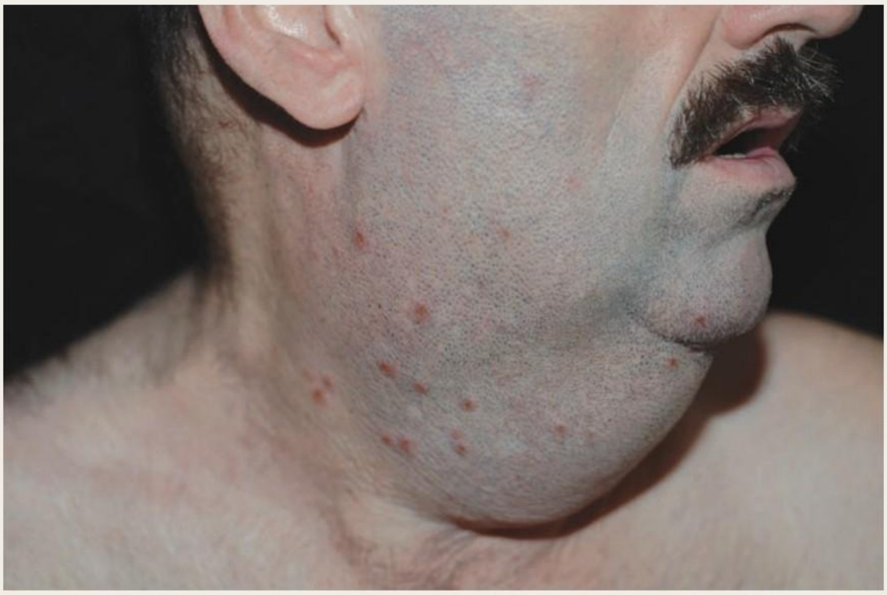

Figure 31-27. Infectious folliculitis A male patient with HIV/AIDS and persistent pruritic pustular and ruptured lesions on the cheek/beard for several months.

Hot-Tub Folliculitis *(Pseudomonas Aeruginosa).* Occurs on the trunk following immersion in spa water (Fig. 31-28).

Dermatophytic Folliculitis. Infection begins in the perifollicular stratum corneum and spreads into follicular ostia and hair shafts (see Section 26) (Fig. 31-29).

Tinea Capitis (see Section 26).

In dermatophytic Majocchi granuloma, scattered papules, pustules, and nodules, usually associated with tinea cruris or tinea corporis.

***Malassezia* Folliculitis.** More common in subtropical and tropical climates. Pruritic, monomorphic eruption characterized by follicular papules and pustules on the trunk, most often on the back (Fig. 31-30), upper arms, and less often on the neck and face; excoriated papules. Absence of comedones differentiates it from acne vulgaris (see Section 26). *Synonym*: Pityrosporum folliculitis.

Candida Albicans. Occurs in sites of occluded skin such as the back of a hospitalized febrile patient or under plastic dressing, especially if topical glucocorticoid preparations are used (see Section 26).

Herpetic Folliculitis. Occurs predominantly in the beard area (viral sycosis) in men. Characterized by follicular vesicles and later crusts (Fig. 31-31).

Molluscum Folliculitis. Presents as umbilicated skin-colored papules in a follicular and perifollicular distribution over the beard area.

Syphilitic (Luetic) Folliculitis: Secondary. Nonscarring alopecia of the scalp and beard (alopecia areolaris); "moth-eaten" appearance.

Demodicidosis. Clinical presentation: perifollicular scaling (pityriasis folliculorum) or rosacea-like erythematous follicular papules and pustules with a background of erythema on the face. Etiology: *Demodex folliculorum.*

Differential Diagnosis

Follicular Inflammatory Disorders. Acneiform disorders (acne vulgaris, rosacea, perioral dermatitis), HIV-associated eosinophilic folliculitis, chemical irritants (chloracne), acneiform adverse cutaneous drug reactions [epidermal growth factor receptor inhibitors (e.g., erlotinib), halogens, glucocorticoids, lithium], keloidal folliculitis, pseudofolliculitis barbae.

Regional Differential Diagnosis. *Face*: acne, rosacea, perioral dermatitis, keratosis pilaris, pseudofolliculitis barbae (ingrowing hairs). *Scalp*: folliculitis necrotica. *Trunk*: acne vulgaris, on the back (Fig. 31-30), upper arms, and less often on the neck and face; excoriated papules. Absence of comedones differentiates it from acne vulgaris (see Section 26). *Synonym*: Pityrosporum folliculitis.

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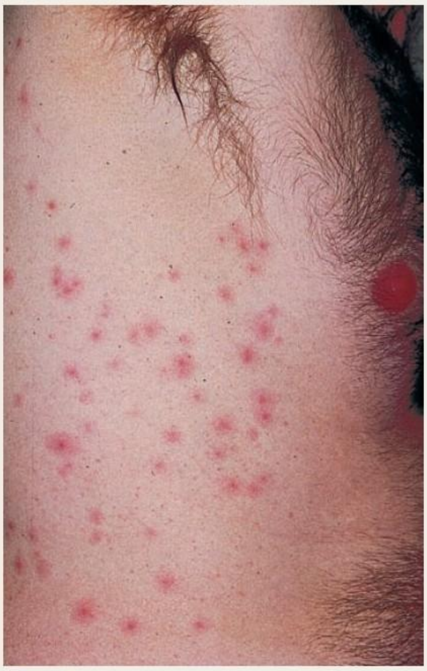


Figure 31-28. Infectious folliculitis ("hot tub"): *P. aeruginosa* A 31-year-old male with multiple painful follicular pustules 3 days after bathing in a hot tub. *P. aeruginosa* was isolated on culture from a lesion.

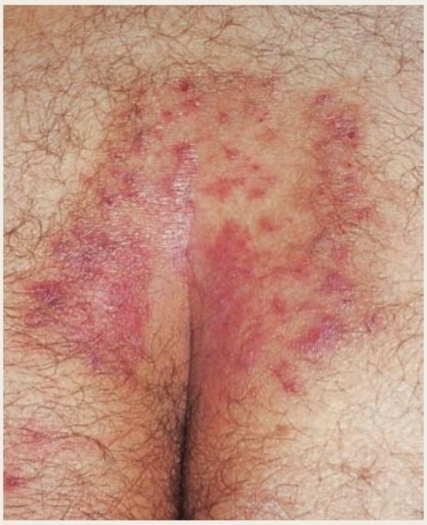


Figure 31-29. Dermatophytic folliculitis: *Trichophyton rubrum* A 31-year-old male with HIV/AIDS had a pruritic rash on the buttocks for 1 year; topical glucocorticoids and antifungal agents had not been effective. Multiple follicular papules and scaling erythema are seen on the sacral area; tinea cruris and pedis were also present. KOH preparation showed septated hyphae. The lesions resolved with oral terbinafine.

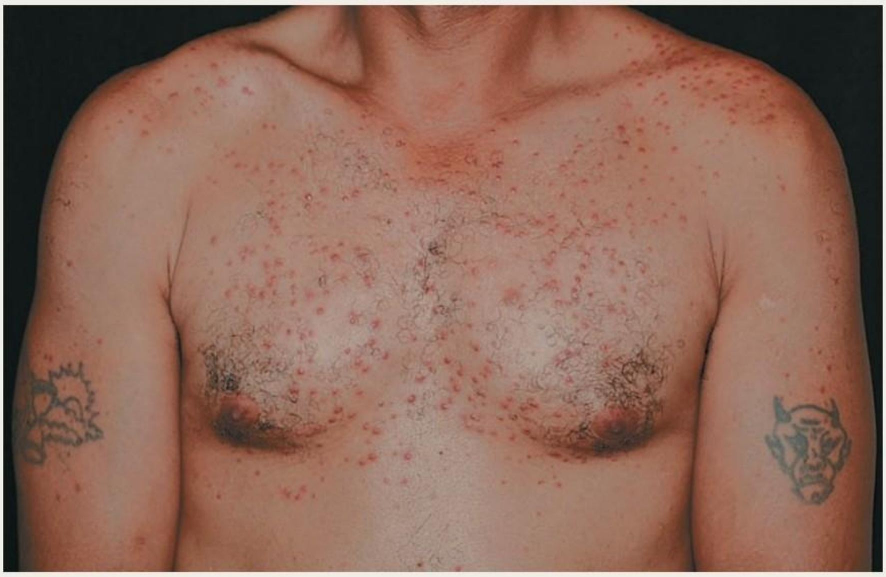


Figure 31-30. Infectious folliculitis: *Malassezia furfur* A 41-year-old Hispanic male with multiple, discrete, follicular papulopustules on the chest.

out pilosebaceous involvement/destruction,

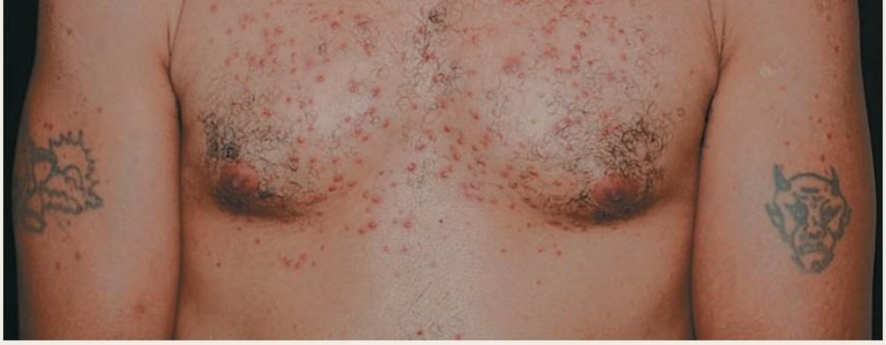


Figure 31-30. **Infectious folliculitis: *Malassezia furfur*** A 41-year-old Hispanic male with multiple, discrete, follicular papulopustules on the chest.

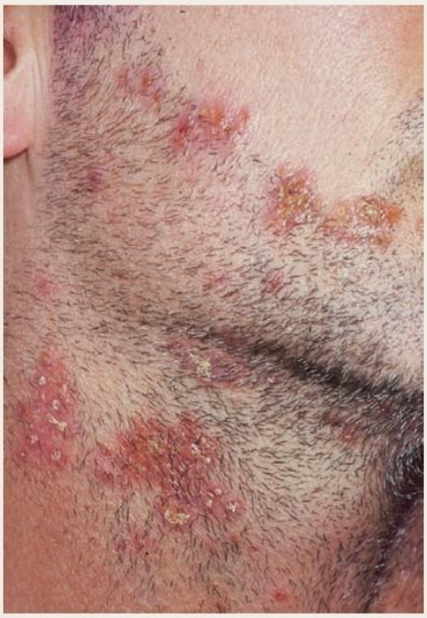


Figure 31-31. **Infectious folliculitis: herpes simplex virus** A 40-year-old healthy male with discrete and grouped pustules and erosions in the beard area for 3 weeks. Lesions resolved with oral acyclovir.

pustular miliaria, transient acantholytic disease (Grover disease). *Axillae and groins*: hidradenitis suppurativa.

Laboratory Findings

Direct Microscopy. *Gram stain S. aureus:* gram-positive cocci. Also visualizes fungi.
KOH Preparation. Dermatophytes: hyphae, spores. *M. furfur*: multiple yeast forms; *Candida*: mycelial forms.
Culture Bacterial. *S. aureus, P. aeruginosa*; gram-negative folliculitis: *Proteus, Klebsiella, Escherichia coli*. In cases of chronic relapsing folliculitis, culture nares and perianal region for *S. aureus* carriage.
Fungal. Dermatophytes; *C. albicans*.
Viral. Herpes simplex virus.
Dermatopathology. Follicular and perifollicular infiltrate which may be lymphocytic (viral, fungal), neutrophilic (bacterial, fungal), granulomatous (viral, fungal) or mixed, with or without pilosebaceous involvement/destruction, Gram stain and fungal stains may be necessary to highlight microorganisms.

Diagnosis

Clinical findings confirmed by laboratory findings.

Course and Prognosis

- *S. aureus* folliculitis can progress to deeper follicular and perifollicular infection with abscess (furuncle, carbuncle) or cellulitis.
- Infection of multiple contiguous follicles results in a carbuncle.
- Many types of infectious folliculitis tend to recur or become chronic unless the predisposing conditions are corrected.

Management

Prophylaxis. *Correct underlying predisposing condition*. Washing with antibacterial soap or benzoyl peroxide preparation or isopropyl/ethanol gel.
Antimicrobial Therapy. **Bacterial Folliculitis.** Most will respond to natural penicillins but can consider dicloxacillin, amoxicillin, primary cephalosporins and clindamycin, usually for 7 to 10 days. Consider culture for resistant organisms. Minocycline, trimethoprim-sulfamethoxazole and quinolones may be necessary. There may be higher resistance to the erythromycin family.
Gram-Negative Folliculitis. Associated with systemic antibiotic therapy of acne vulgaris. Discontinue current antibiotics. Wash with benzoyl peroxide. In some cases, ampicillin (250 mg four times daily) or trimethoprim–sulfamethoxazole four times daily. Isotretinoin.
Fungal Folliculitis. Various topical antifungal agents. For dermatophytic folliculitis: terbinafine, 250 mg po for 14 days, or itraconazole, 100 mg twice daily for 14 days. For *Candida* folliculitis: fluconazole or itraconazole, 100 mg twice daily for 14 days.
Herpetic Folliculitis. See "Herpes Simplex Virus Infections" in Section 27.
Demodicidosis. Permethrin cream. Ivermectin, 200 μg/kg (usual range, 12–18 mg).
Pseudofolliculitis Barbae. Rule out secondary *S. aureus* infection. Discontinue shaving. Use beard clipper instead of safety razor. Destruction of hair follicle: electrolysis; laser hair removal.

Figure 32-6. Habit-tic deformity The nail plates of both thumbs are dystrophic with transverse ridging and discoloration. The cuticle is absent and the proximal nail folds excoriated. When the proximal nails and nail fold were covered with tape continually, normal nails regrew in 5 months.

Figure 32-7. Compulsive nail picking The cuticles are not formed, the proximal nail folds are inflamed and excoriated. The breaks in the integrity provide a portal of entry for *S. aureus* and acute paronychia.

Nail Apparatus Involvement of Cutaneous Diseases

Psoriasis ☐ ◐

- Most common dermatosis affecting the nail apparatus.
- >50% of persons with psoriasis have nail involvement at one point in time, with lifetime involvement up to 80–90%.
- See also "Psoriasis" in Section 3.

Laboratory Examination

KOH preparation and/or nail clipping to pathology for PAS stain to rule out fungal colonization/infection. Onychomycosis is more common in nails with onycholysis.

Clinical Findings

Skin. Typical psoriatic lesion on nail folds (Fig. 32-8).

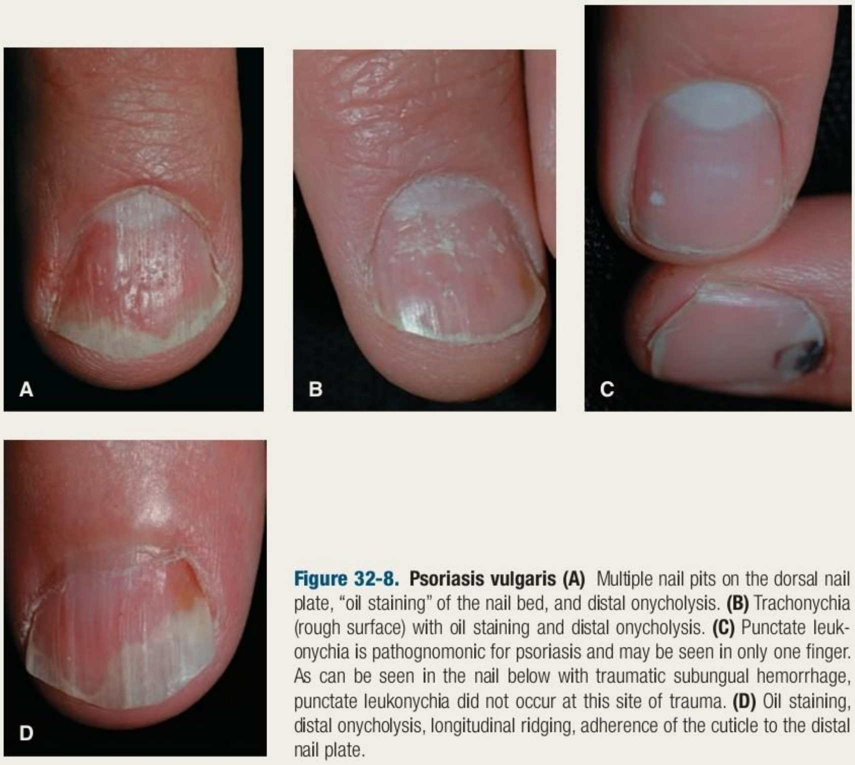


Figure 32-8. Psoriasis vulgaris (A) Multiple nail pits on the dorsal nail plate, "oil staining" of the nail bed, and distal onycholysis. **(B)** Trachonychia (rough surface) with oil staining and distal onycholysis. **(C)** Punctate leukonychia is pathognomonic for psoriasis and may be seen in only one finger. As can be seen in the nail below with traumatic subungual hemorrhage, punctate leukonychia did not occur at this site of trauma. **(D)** Oil staining, distal onycholysis, longitudinal ridging, adherence of the cuticle to the distal nail plate.

Matrix

- *Pitting or elkonyxis*: Punctate depressions; small, shallow; vary in size, depth, shape (Fig. 32-8A). May occur as regular lines (transverse; long axis) or grid-like pattern. Uncommon on toenails. Also seen in atopic dermatitis. Geometric and superficial pits seen in alopecia areata (hammered brass nails).
- *Trachyonychia*: Nail dull, rough, fragile (Fig. 32-8B). Twenty-nail dystrophy or sandpaper nails associated with proximal nail matrix damage: nonspecific and can also be seen in alopecia areata (see Fig. 32-10), lichen planus, atopic dermatitis. May regress spontaneously.
- *Serial transverse depressions*: May mimic "washboard" nails of tic habit (pushing back
- *Punctate leukonychia*: 1- to 2-mm white spots in nail plate (mistakenly attributed to trauma) (Fig. 32-8C).
- *Leukonychia*: Proximal matrix involvement: surface rough and nail coarse (Fig. 32-8C).

Nail Bed

- *"Oil" spots*: Oval, salmon-colored nail beds (Fig. 32-8A, D).
- *Onycholysis*: Secondary to "oil" spots affecting hyponychium medially or laterally (Fig. 32-8A). May become colonized with *Candida*, environmental fungi (e.g., *Aspergillus*), *Pseudomonas*. Predisposes to distal/lateral onychomycosis in toenail. Up to 20% of psoriatic nails have secondary onychomycosis.
- *Subungual hyperkeratosis*: Nail plate becomes

plate evolving into distal split nail (onychorrhexis) (Fig. 32-9B).
- *Diffuse matrix involvement*: Selective atrophy of nail plate with onychorrhexis and/or transverse splitting.
- *Red lunula*: Focal or disseminated.
- *Melanonychia, longitudinal*: Transitory.
- *Complete nail split.*
- *Pterygium formation (scar, matrix destroyed)*: Partial loss of the central nail plate presents as a V-shaped extension of skin of proximal nail fold adherent to nail bed (Fig. 32-9A, B).
- *20-nail dystrophy of childhood*: Resolves spontaneously.
- *LP-like eruptions following bone marrow transplant*: Graft-versus-host disease.
- *Drug-induced LP-like reaction*.

Management

- See "Lichen Planus," Section 14.
- Intralesional triamcinolone.
- Systemic glucocorticoids.

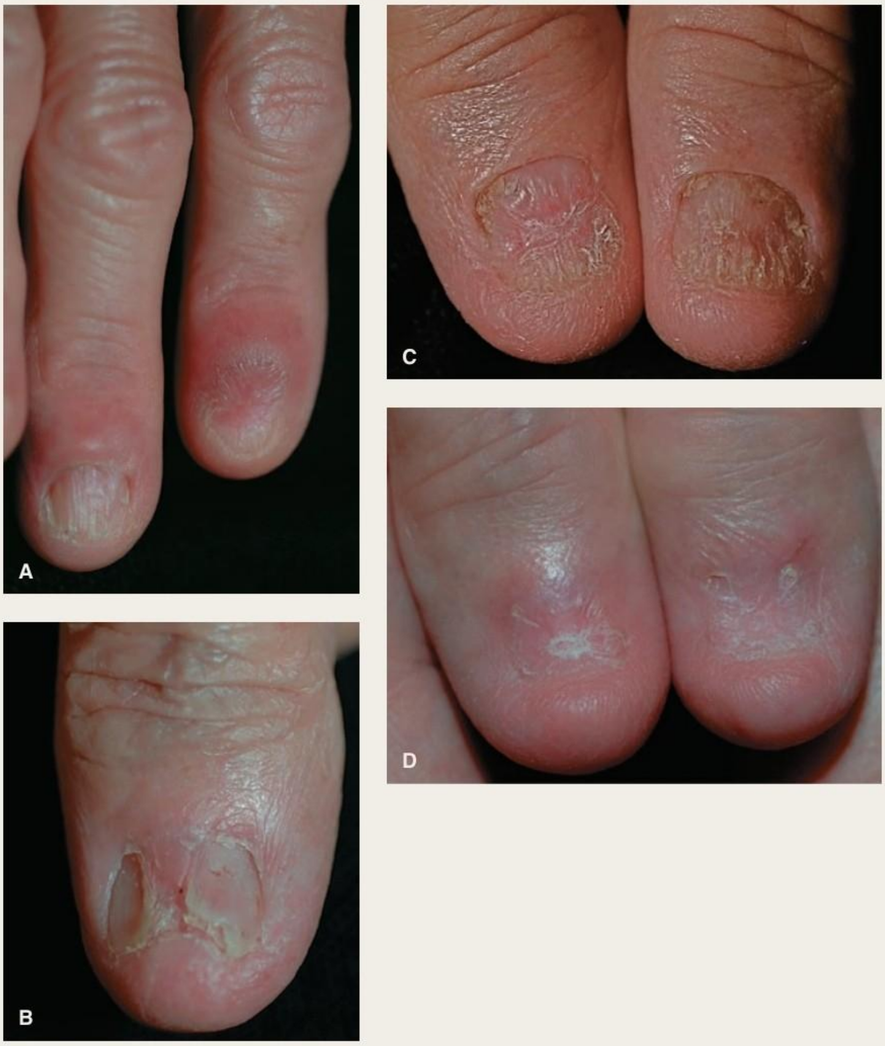


Figure 32-9. Lichen planus (A) Middle finger: involvement of the proximal fold and matrix has caused trachonychia, longitudinal ridging, and pterygium formation. Index finger: destruction of the matrix and nail plate is complete with anonychia. Seven of ten fingernails are involved; the others are normal. **(B)** Involvement of the nail matrix with scarring or pterygium formation proximally dividing the nail plate in two. **(C)** Early involvement of the matrix with thinning of the thumbnail plates. **(D)** Same patient as Fig. 32-8C 2 years later, the nail plate is completely destroyed, i.e., anonychia.

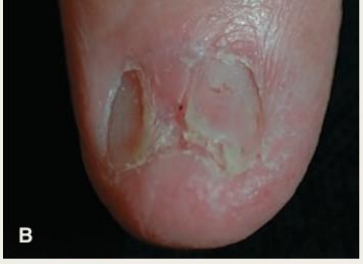


Figure 32-9. Lichen planus (A) Middle finger: involvement of the proximal fold and matrix has caused trachonychia, longitudinal ridging, and pterygium formation. Index finger: destruction of the matrix and nail plate is complete with anonychia. Seven of ten fingernails are involved; the others are normal. **(B)** Involvement of the nail matrix with scarring or pterygium formation proximally dividing the nail plate in two. **(C)** Early involvement of the matrix with thinning of the thumbnail plates. **(D)** Same patient as Fig. 32-8C 2 years later, the nail plate is completely destroyed, i.e., anonychia.

Alopecia Areata (AA)

- See “Nonscarring Alopecia,” Section 31.
- Manifestations:
    - Geometric pitting (Fig. 32-10) (small, superficial, regularly distributed).
- Hammered brass appearance.
- Mottled erythema of lunulae.
- Trachonychia (roughness caused by excessive longitudinal striations).

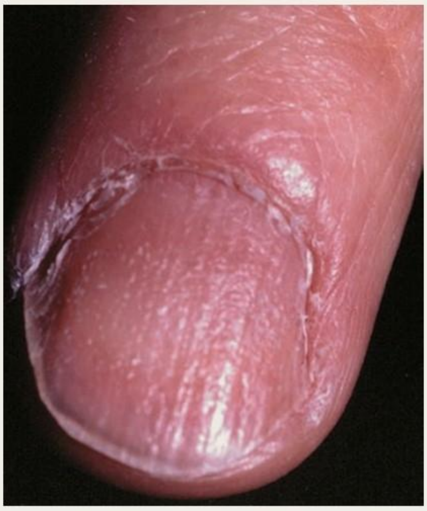


Figure 32-10. Alopecia areata: trachonychia The nail plate is rough with a “hammered brass” appearance.

Darier Disease (Darier–White Disease, Keratosis Follicularis)

Nail changes are pathognomonic: longitudinal streaks (red and white); distal subungual hyperkeratotic papules with distal V- or wedge-shaped fissuring of nail plate (Fig. 32-11).

Acute Paronychia ICD-10 ◦ L03.01

- Acute infection of lateral or proximal nail fold.
- Usually associated with break in integrity of epidermis (e.g., hang nail), trauma.
- *Findings*: Throbb
± abscess forma
- Infection may ex
(Fig. 32-18).

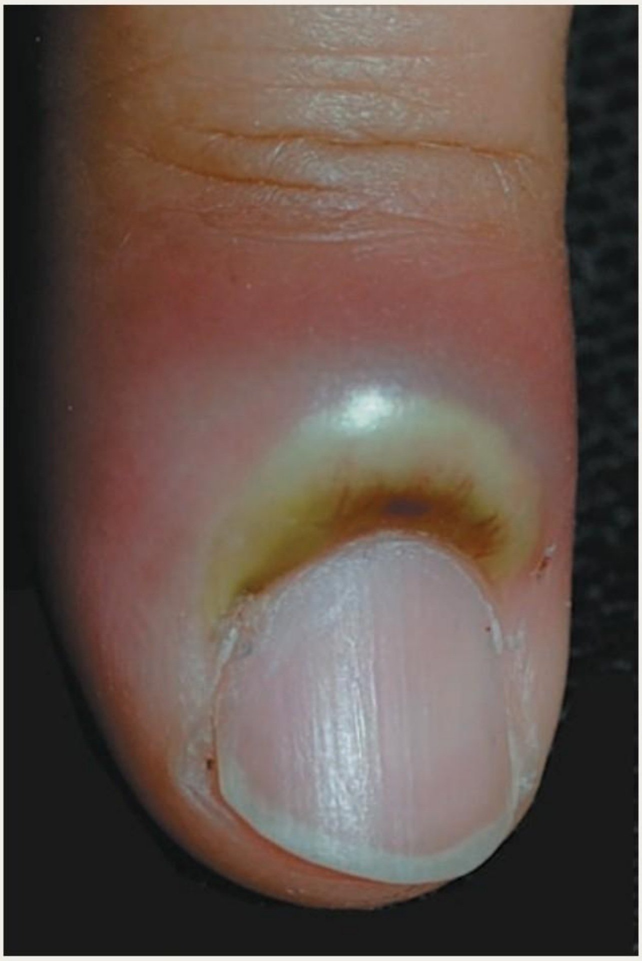


Figure 32-17. Acute paronychia The proximal nail fold is red and edematous (cellulitis) with pus formation.

Figure 32-18. Fel
tip with surrounding
sensitive *S. aureus*
the pus.

Felon ICD-9: 681.01 ◦ ICD-10: L03.0

- Soft-tissue infection of pulp space of distal phalanx (Fig. 32-18); closed space infection of multiple
- *Complications*: C
phalanx, seques

s and Onychomycosis

sually cause "space" infec-
paronychia or onycholytic
se destruction of the nail in
promised host.
infect the skin around the
d cause superficial destruc-

- *Onychomycosis*: Chronic progressive fungal infection of nail apparatus, most commonly caused by dermatophytes, less often by *Candida* spp.; molds and environmental fungi can be cultured from diseased nails but are not usually primary pathogens.

chia

☐ ◐

infections of the nail apparatus
on fingers, commonly as
n of chronic paronychia. Onychia
ation of the matrix of the nail
ing of nail.

- Invasion of nail plate usually occurs only in the immunocompromised host, i.e., chronic mucocutaneous candidiasis (CMC) or HIV/AIDS disease.

demiology

and other species. Normal
infection if local ecology is
yeast or in association with
tus. See "Candidiasis," Sec-

tion associated with ony-

es of chronic paronychia.
tinea unguium.
ophy (Fig. 32-19): chronic
IDS disease.

Candidiasis," Section 26.

s

Section 26.
l onychia and paronychia
ldren with HIV/AIDS, often
ıcosal candidiasis.
ronic Paronychia with Acute
dida spp. can cause painful
with pain, tenderness, ery-
nay become dystrophic with
on; white, yellow, green, or
with transverse furrowing.
Unguium. Secondary patho-
onychomycosis.
Proximal/lateral nail folds
hickened. Fingertips appear
ıvaded and may eventually
strophic (Fig. 32-19). HIV/
be involved. CMC: 20 nails
time.
lidiasis," Section 26.

Differential Diagnosis

Tinea unguium, psoriasis, eczema, chronic paronychia, lichen planus.

Management

See "Candidiasis," Section 26.

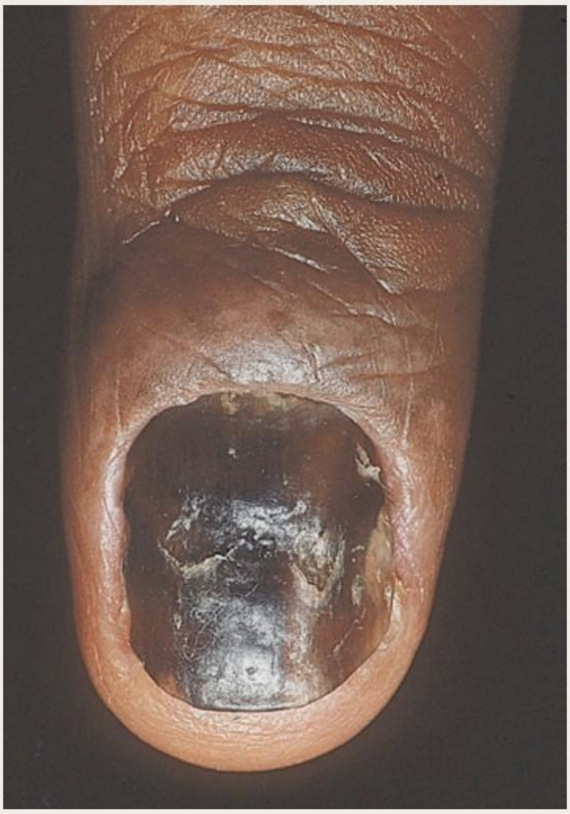


Figure 32-19. ***Candida*** **onychomycosis: total dystrophic type** The entire fingernail plate is thickened and dystrophic and is associated with a paronychial infection; both findings were caused by *C. albicans* in an individual with advanced HIV/AIDS disease.

Distal and Lateral Subungual Onychomycosis (DLSO) (Fig. 32-20). Infection begins in hyponychial area or nail fold, extending subungually. May be either primary, i.e., involving a healthy nail apparatus, or secondary (e.g., psoriasis) associated with onycholysis. Always associated with tinea pedis.

Superficial White Onychomycosis (SWO). Pathogen invades surface of dorsal nail (Fig. 32-21). *Etiology*: *Trichophyton mentagrophytes* or *T. rubrum* (children). Much less commonly, mold: *Acremonium*, *Fusarium*, *Aspergillus terreus*.

Proximal Subungual Onychomycosis (PSO). Pathogen enters by way of the posterior nail fold–cuticle area and then migrates along the proximal nail groove to involve the underlying matrix, proximal to the nail bed, and finally the underlying nail (Fig. 32-22). *Etiology*: *T. rubrum*. *Findings*: Leukonychia that extends distally from under proximal nail fold. Usually one or two nails involved. Always associated with immunocompromised states.

Age of Onset. Children or adults. Once acquired, usually does not remit spontaneously. Therefore, the incidence increases with advancing age; 1% of individuals <18 years affected; almost 50% of those >70 years.

Sex. Somewhat more common in men.

Etiologic Agents. Between 95% and 97% caused by *T. rubrum* and *T. mentagrophytes*.

Molds. *Acremonium, Fusarium*, and *Aspergillus* spp. can rarely cause SWO. Dermatosis such as psoriasis, which results in onycholysis and subungual hyperkeratosis, or dermatophytic onychomycosis can be secondarily colonized/infected by molds.

Geographic Distribution. Worldwide. Etiologic agent varies in different geographic areas. More common in urban than in rural areas (associated with wearing occlusive footwear).

Prevalence. Incidence varies in different geographic regions. In the United States and Europe, up to 10% of adult population affected (related to occlusive footwear). In developing nations where open footwear is worn, uncommon.

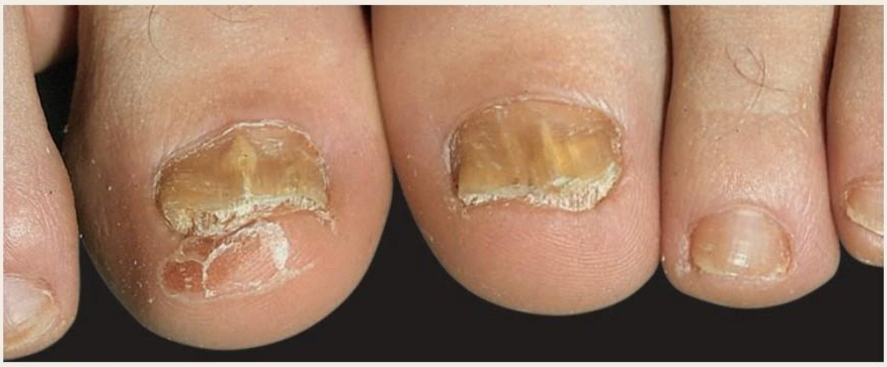


Figure 32-20. Onychomycosis of toenails: distal and lateral subungual type (DLSO) The toenails are white, caused by onycholysis and subungual hyperkeratosis. The dorsum of the feet shows erythema and scaling, i.e., tinea pedis. *T. rubrum* was detected on culture.

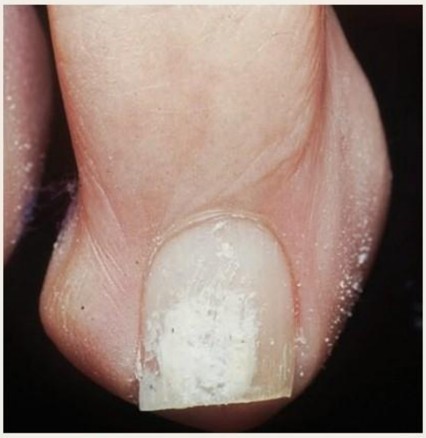


Figure 32-21. Onychomycosis of toenails: superficial white type (SWO) The dorsal nail plate is chalky white. White nail dystrophy can easily be treated by curettage; KOH preparation of the curetting shows hyphae.

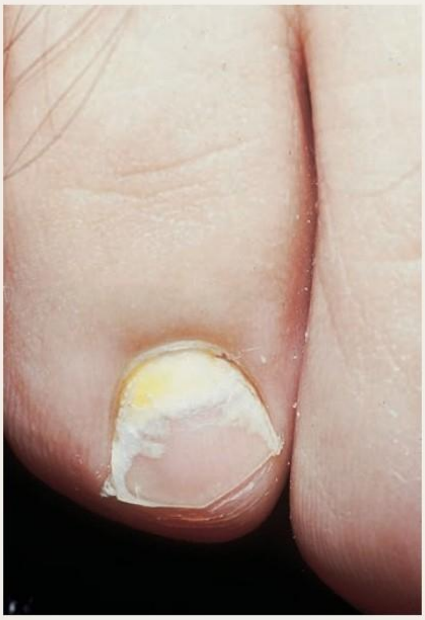


Figure 32-22. ***Tinea unguium*: proximal subungual onychomycosis type** The proximal nail plate is a chalky white color due to invasion from the undersurface of the nail matrix. The patient had advanced HIV/AIDS disease.

Transmission. **Dermatophytes**. Anthropophilic dermatophyte infections are transmitted from one individual to another, by fomite or direct contact, commonly among family members. Some spore forms (arthroconidia) remain viable and infective in the environment for up to 5 years.

Molds. Ubiquitous in environment; not transmitted between humans.

Risk Factors. Atopics are at increased risk for *T. rubrum* infections. Diabetes mellitus, treatment with immunosuppressive drugs, HIV/AIDS. For toenail onychomycosis, most im-

of normal nail plate remains unimpaired despite fungal infection.

Clinical Manifestation

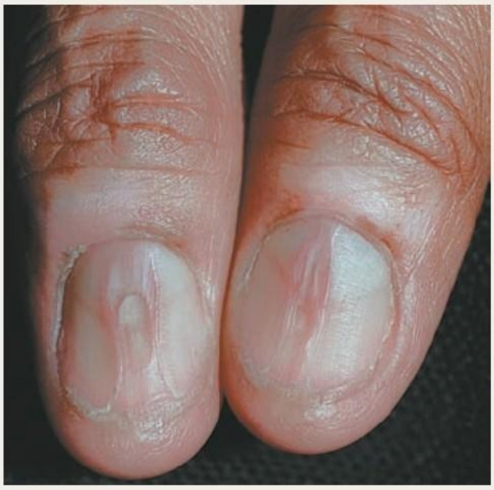


Figure 32-29. Discoid lupus erythematosus: Nail fold and matrix involvement and nail dystrophy Proximal nail folds show erythema, scarring, and depigmentation associated with nail matrix inflammation.

Pterygium Inversum Unguium

Nail plate adheres to fingertip skin in scleroderma.

Systemic Amyloidosis

Nail dystrophy resembling lichen planus with severe onychodystrophy (nail plate thinned, longitudinally fissured with subungual hemorrhages) can precede diagnosis of primary systemic amyloidosis. Biopsy of nail apparatus confirms the diagnosis of amyloidosis with amyloid deposits in the superficial dermis of the nail matrix (Fig. 32-30).

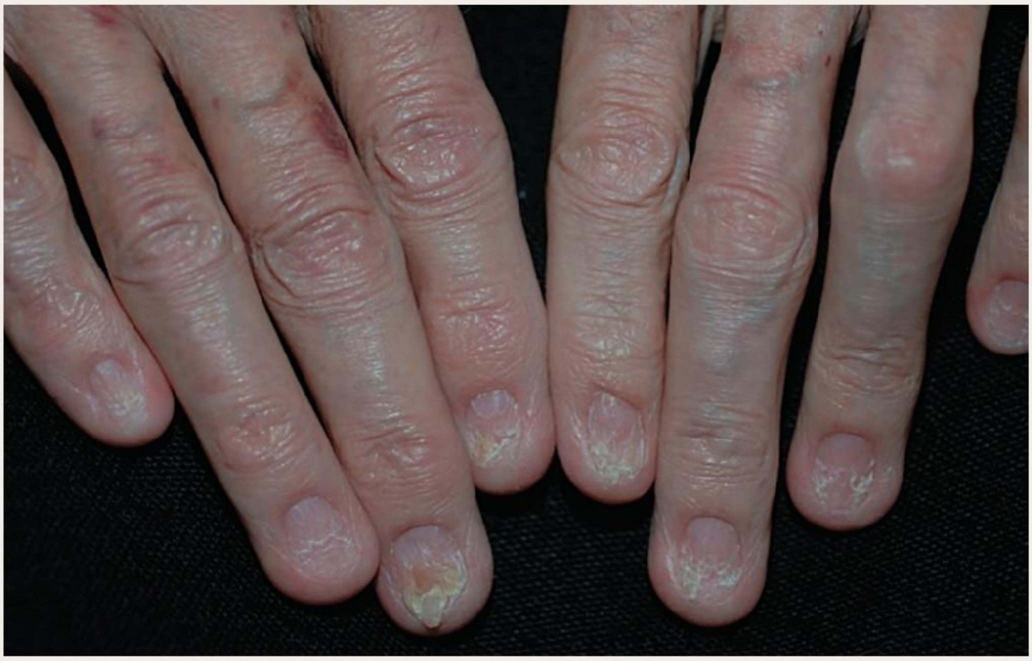


Figure 32-30. Systemic amyloidosis Nail findings preceded the diagnosis of systemic amyloidosis. The matrix is inflamed with resultant thinning of the proximal nail plate and disintegration distally.

es of the Lips ICD-9: 528.5 ◦ ICD-10: K13.0

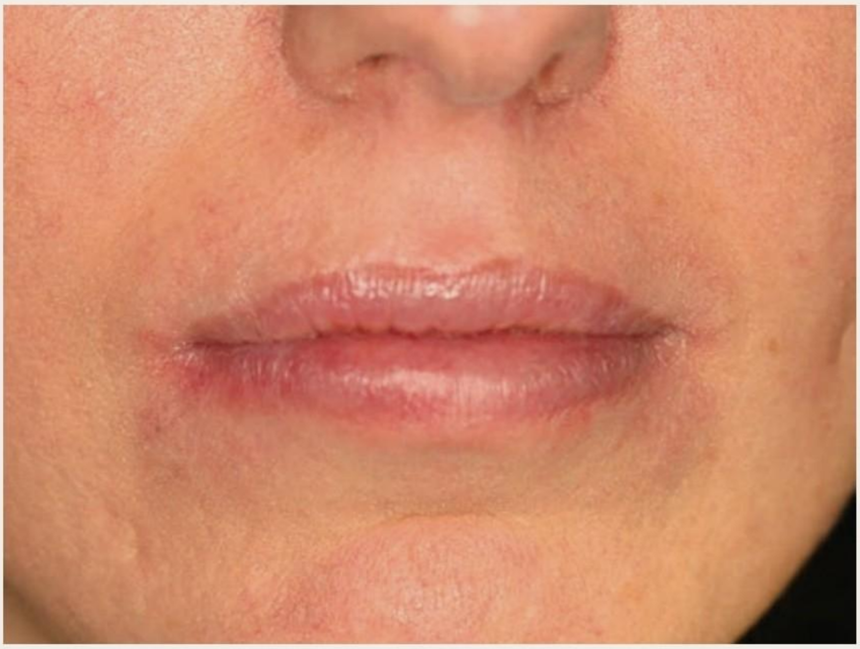
r Cheilitis (Perlèche) ☐ ◐

ited with increased moisture at
ssures, salivation (at sleep).

posing factors: thumb sucking in children;
 face and loss of teeth in older persons;
asis in immunocompromised persons;
lococcus aureus in atopic dermatitis and
noin treatment.

- *Findings*: erythema and maceration at commissures (see Fig. 33-1); white candidal colony.
- *Diagnosis*: KOH for candidiasis; culture for *S. aureus, Candida*.
- *Management*: Identify and treat causes.

. Angular chei-
thema and scal-
al commissures.
Dr. Nathaniel Tre-



Cheilitis ☐ ◐

r keratoses, usually of the lower lip.
uamous cell carcinoma in situ (SCCIN) or invasive if papule or nodule or ulcer occurs. (See "Solar Keratosis" in Section 10.)

ions of the Tongue, Palate, and Mandible
6, 528.7, 529 ◦ ICD-10: K14

ed Tongue ☐ ●

variant in up to 11% of population.
omatic.
s: Multiple folds with anterior-posterior

- *Associated disorders*: Psoriasis, Down syndrome, acromegaly, Sjögren syndrome.
- *Synonyms*: Lingua fissurata, lingua plicata, scrotal

Lichen Planus ◧ ◐

- *Incidence*: 40–60% of individuals with LP have oropharyngeal involvement.
- *Findings*:
  - Milky-white papules.
  - Wickham striae: Reticulate (netlike) patterns of lacy-white hyperkeratosis [buccal mucosa (Fig. 33-6), lips, tongue, and gingivae].
  - Hypertrophic LP—leukoplakia with Wickham striae usually on the buccal mucosa.
  - Atrophic LP—shiny plaque often with Wickham striae in surrounding mucosa.
  - Erosive/ulcerative LP—superficial erosions with overlying fibrin clots that are seen on the tongue and buccal mucosa; can be painful (Fig. 33-6).
  - Bullous LP—intact blisters (rupture and result in erosive LP).
  - Desquamative gingivitis—bright red gingiva (Fig. 33-7).

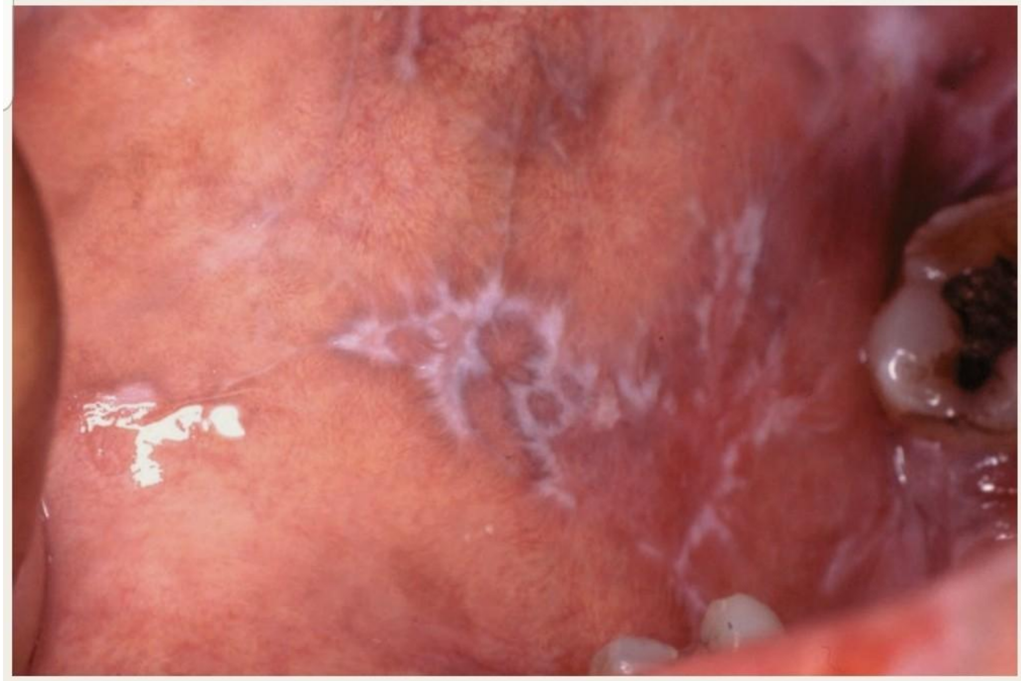


Figure 33-6. Lichen planus: Wickham striae Poorly defined violaceous plaque with lacy, white pattern on the buccal mucosa.

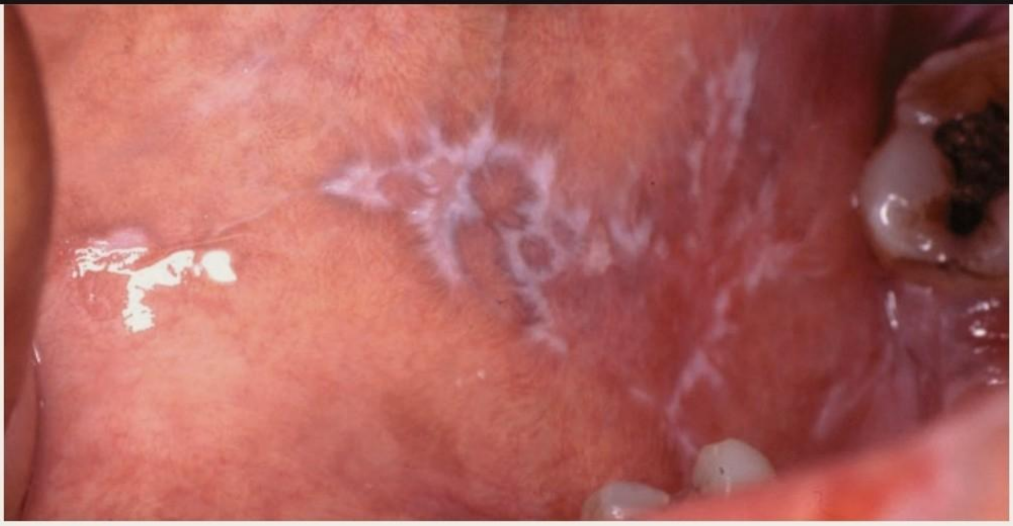


Figure 33-6. Lichen planus: Wickham striae Poorly defined violaceous plaque with lacy, white pattern on the buccal mucosa.

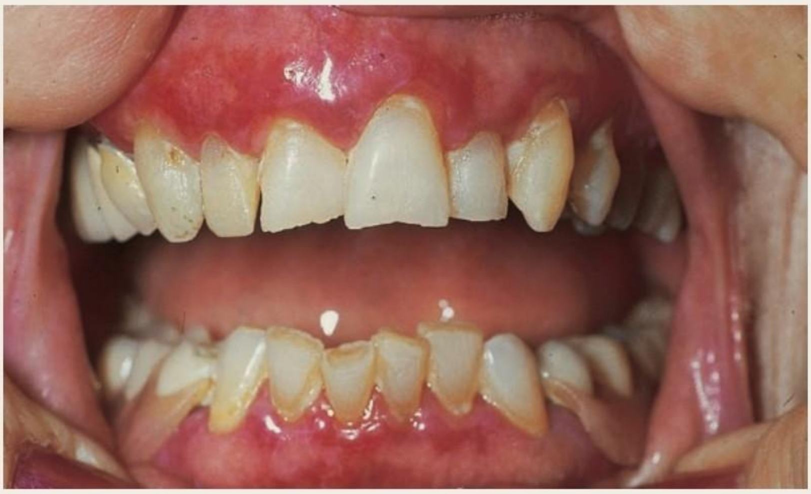


Figure 33-7. Lichen planus: desquamative gingivitis The gingival margins are erythematous, edematous, and retracted. The lesions were painful, making dental hygiene difficult, resulting in plaque formation on the teeth.

Acute Necrotizing Ulcerative Gingivitis ■ ◐

- *Precipitating factors*: Poor oral hygiene, HIV/AIDS, immunosuppression, alcohol and tobacco use, nutritional deficiency.
- *Findings* (Fig. 33-8): Punched-out ulcers of the interdental papillae. Gingival hemorrhage, severe
- *Etiologic agents*: *Bacteroides fusiformis, Prevotella intermedia, Borrelia vincentii, Treponema.*
- *Management*: Systemic antibiotics such as clindamycin, metronidazole, amoxicillin. Dental hygiene.

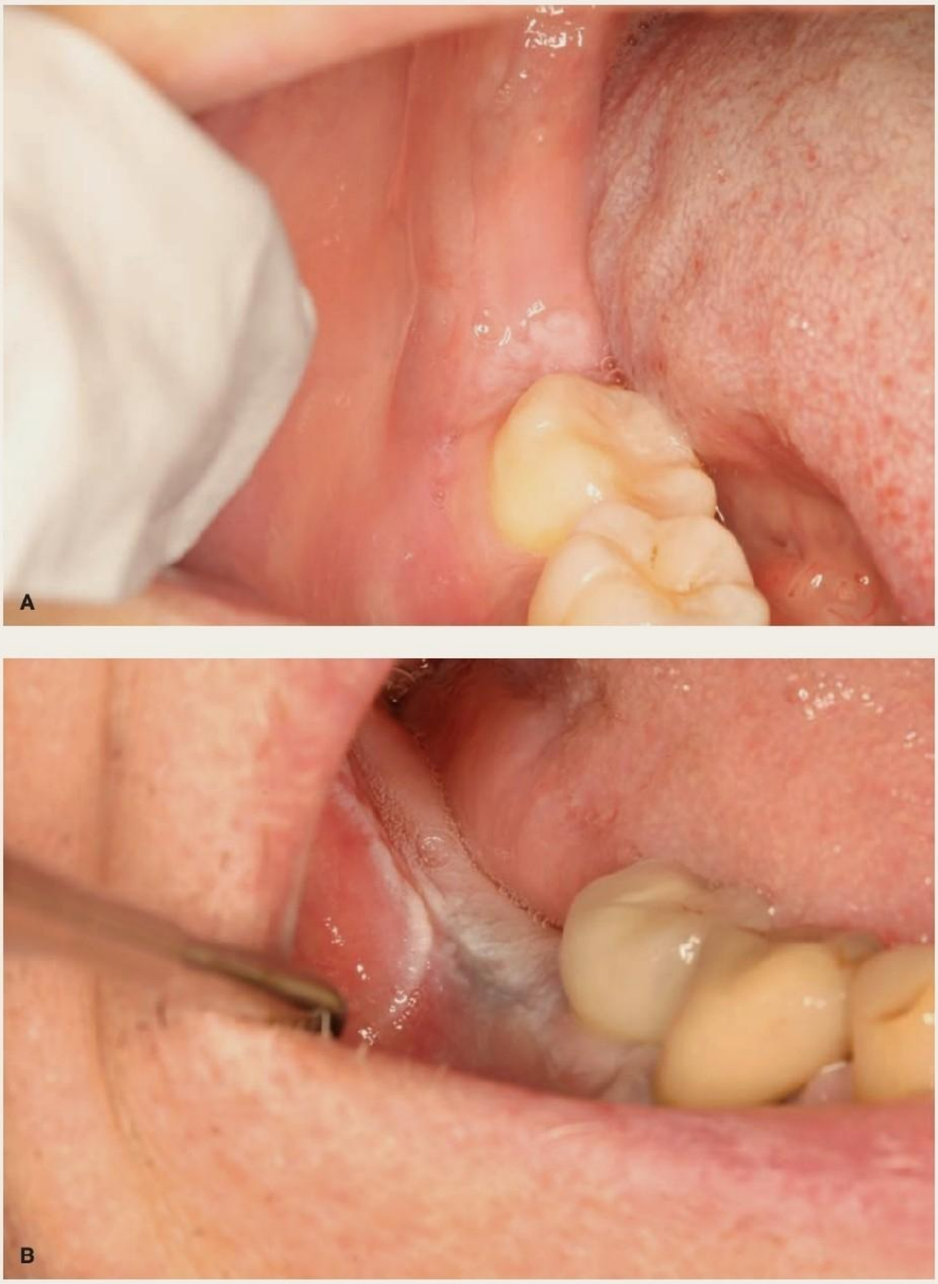
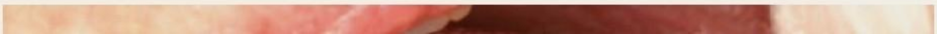


Figure 33-13. **(A, B) Lichen simplex chronicus** Note the white plaque in the retromolar pad (after third molar extractions). These are often seen on edentulous ridge after extractions. (Courtesy of Dr. Sook-Bin Woo.)



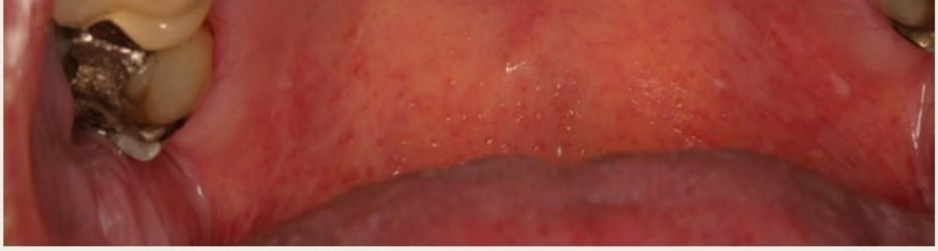


Figure 33-15. Nicotine stomatitis Posterior palate shows erythematous pinpoint papules at sites of ducts, where chemical irritation has caused chronic inflammation. (Courtesy of Dr. Sook-Bin Woo.)

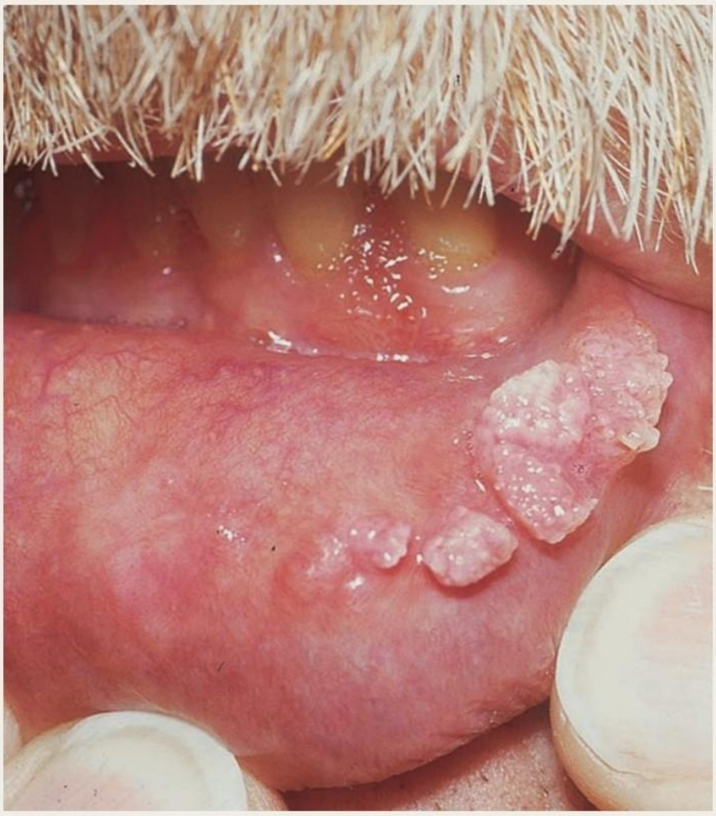


Figure 33-16. Condyloma acuminatum: mucosal lip Cluster of white cauliflower-floret-like lesions on the mucosa of the lower lip.

Erythematous Lesions and/or Leukoplakia ◧ ◐ → ○

- Erythematous lesions ± leukoplakia appear red because of inflammation, hemorrhage, increased angiogenesis, epithelial atrophy, acantholysis, ulceration.
- The differential diagnosis includes SCCIS, invasive SCC, candidiasis, migratory glossitis, radiotherapy and chemotherapy-induced mucositis, lichen planus, lupus erythematosus.

Vitiligo and Leukoderma (See also Section 13) ◧ ●

- *Etiology*: Loss of melanocytes results in depigmentation.
- *Isomorphic* or *Koebner phenomenon*: Depigmentation at sites of injury: genital herpes, cryosurgery, imiquimod therapy.
- Wood lamp examination: Differentiates depigmentation from hypopigmentation.
- *Clinical findings*: Sharply demarcated, depigmented, white macules (Fig. 34-9); examine skin for other depigmented areas.
- *Differential diagnosis*: Lichen sclerosus, site of genital herpes; iatrogenic after cryo-, electro-, or laser surgery.

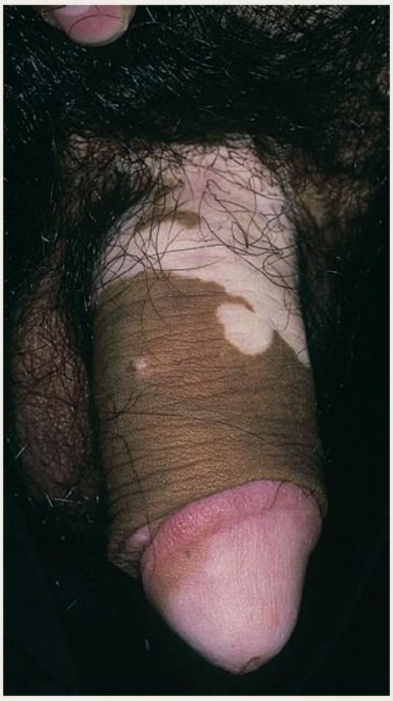


Figure 34-9. Vitiligo: penis Multiple depigmented macules have become confluent.

Psoriasis Vulgaris (See also Section 3) ☐ ●

- *Incidence*: Most common noninfectious dermatosis occurring on the glans penis and vulva.
- *Onset*: May be initial presentation of psoriasis.
- *Clinical findings*: (1) Erythematous scaling plaques on nonoccluded skin (Fig. 34-10); (2) intertriginous psoriasis, well-demarcated erythematous plaques without scale in naturally occluded skin (Fig. 34-11).
- *Distribution [intertriginous (inverse) psoriasis]*: Penis, vulva, intergluteal cleft, inguinal folds.
- *Differential diagnosis*: Lichen planus (LP), fixed drug eruption, condyloma acuminata, HPV-induced IN, squamous cell carcinoma (SCC) *in situ*, invasive SCC, extramammary Paget disease, migratory necrolytic erythema.

- *Onset*: May be initial presentation of psoriasis.
- *Clinical findings*: (1) Erythematous scaling plaques on nonoccluded skin (Fig. 34-10); (2) intertriginous psoriasis, well-demarcated erythematous plaques without scale in naturally occluded skin (Fig. 34-11).
- *Differential diagnosis*: Lichen planus (LP), fixed drug eruption, condyloma acuminata, HPV-induced IN, squamous cell carcinoma (SCC) *in situ*, invasive SCC, extramammary Paget disease, migratory necrolytic erythema.

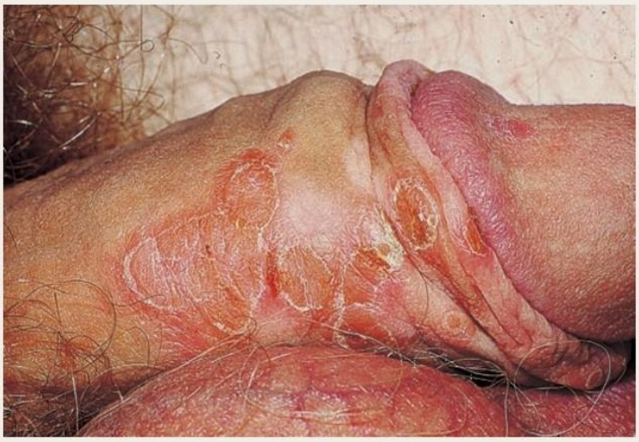


Figure 34-10. Psoriasis vulgaris: shaft of penis Well-demarcated scaling plaques on the penile shaft of a 25-year-old male. “Pinking” of the intergluteal cleft and nail findings of psoriasis were also present. The patient presented to a clinic for sexually transmitted disease.

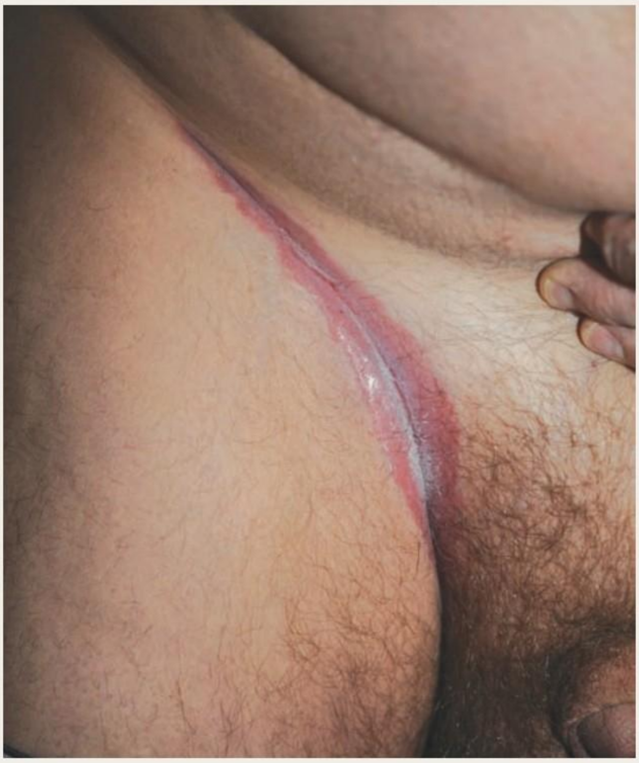


Figure 34-11. Psoriasis vulgaris: intertriginous An erythematous plaque, present for decades and unresponsive to topical antifungal agents, is seen in the right inguinal area. Biopsy excluded extramammary Paget disease.

Lichen Planus (See also Section 14)

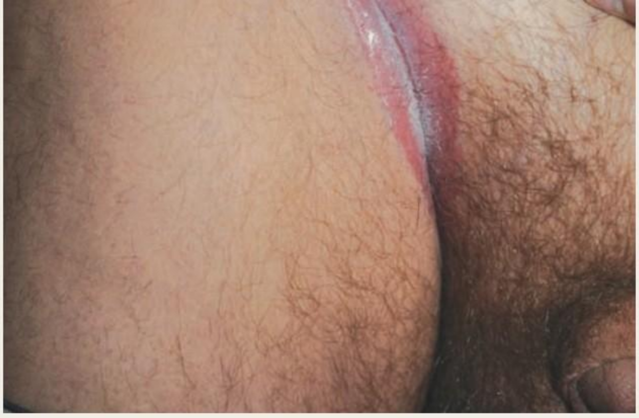


Figure 34-11. Psoriasis vulgaris: intertriginous An erythematous plaque, present for decades and unresponsive to topical antifungal agents, is seen in the right inguinal area. Biopsy excluded extrammary Paget disease.

Lichen Planus (See also Section 14)

- Commonly associated with LP at other sites: however, may occur as initial or sole manifestation.
- *Symptoms*: Not pruritic; pain in eroded lesions, anxiety about sexually transmitted disease.
- *Clinical findings*: Violaceous flat-topped papules, discrete or confluent. Lacy white surface pattern most commonly on glans. Older lesions may have grayish hue with melanin incontinence. Annular lesions occur on glans and shaft (Fig. 34-12). Bullous and/or erosive LP (Fig. 34-13) on glans, vulva.
- *Distribution*: Glans, penile shaft (Fig. 34-12), vulva.
- *Course*: Spontaneous remission; erosive LP may persist for decades; SCC complicates rarely.

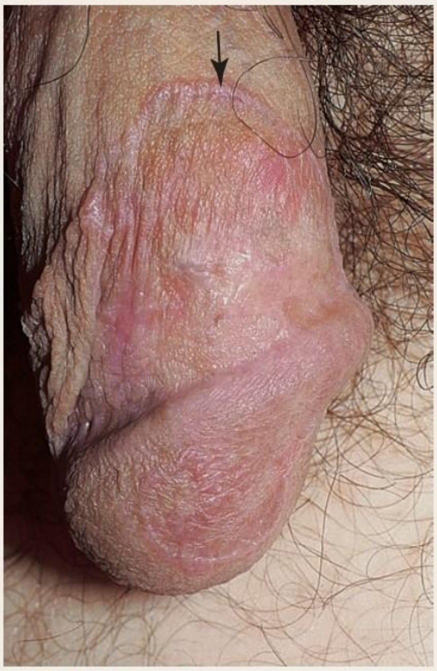


Figure 34-12. Lichen planus, annular: penis Violaceous annular plaques (*arrow*) on the distal shaft and glans of a 26-year-old patient, present for >1 year. White lacelike plaques were also present on the buccal mucosa.

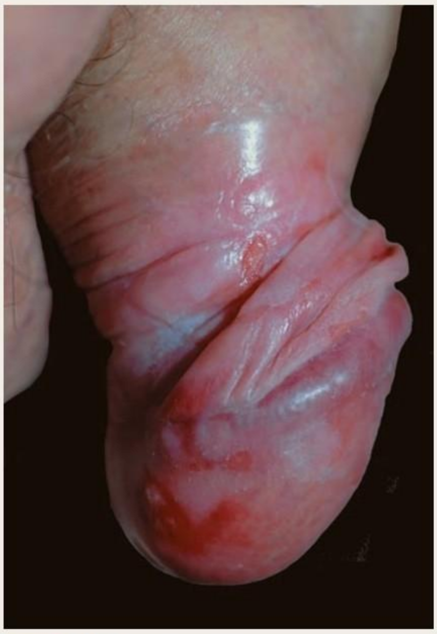


Figure 34-13. Lichen planus, erosive: penis A 36-year-old male with painful erythematous erosions on the glans penis and foreskin for 6 months. Lesions resolved with intralesional triamcinolone injections.

- Most skin eruptions and rashes are more or less pruritic, but there are states where there is severe pruritus in the absence of skin lesions, except for scratch marks (Fig. 35-1). This is called *pruritus sine materia* (from Latin, "itch without physical substrate").
- The diagnostic approach to the patient with generalized pruritus without identifiable skin lesions is a *diagnosis of exclusion.*
- Pruritus is a symptom of skin disease that at the time of examination does not manifest with specific lesions.
- It may be due to an internal organ disease, metabolic and endocrine conditions, or hematologic disease.
- It may be a manifestation of malignant tumors, psychogenic states, or HIV infection; or it may be related to injected or ingested drugs.
- The various causes of pruritus sine materia are listed in Table 35-1, and an algorithm of how to approach a patient with pruritus sine materia is shown in Table 35-2.
- Skin signs may be clinically inapparent, perhaps confined to only circumscribed areas, and this is particularly important with regard to the exclusion of scabies, pediculosis, or conditions such as urticaria factitia.

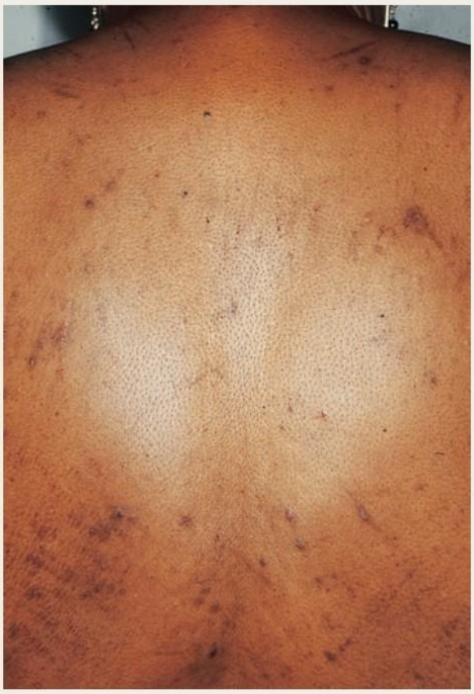


Figure 35-1. Pruritus without diagnostic skin lesions This patient had multiple scratch marks due to compulsive scratching because of severe pruritus. There were no other diagnostic lesions. Workup revealed biliary cirrhosis without jaundice.

Most Important Causes (See Table 35-1)

TABLE 35-1 CAUSES OF PRURITUS SINE MATERIA

- Metabolic, endocrine conditions
  - Hyperthyroidism: probably due to increased blood flow
  - Hypothyroidism: probably due to excessive dryness
  - Pregnancy related
  - Diabetes: pruritus is rarely associated, but can be a symptom of diabetic neuropathy
- Malignant neoplasms: can be the presenting feature
  - Lymphoma, myeloid and lymphatic leukemia, myelodysplasia
  - Multiple myeloma
  - Hodgkin disease
  - Other cancer (rare)
- Drug ingestion
  - Subclinical drug sensitivities
  - Aspirin, alcohol, dextran, polymyxin B, morphine,
  - Codeine, scopolamine, D-tubocurarine,
  - xyethyl starch
- I ons/Infections
  - es[a]
  - Pediculosis corporis, capitis, pubis
  - Hookworm (ancylostomiasis)
  - Onchocerciasis
  - Ascariasis
  - HIV: can be a primary symptom of infection or a chronic comorbidity